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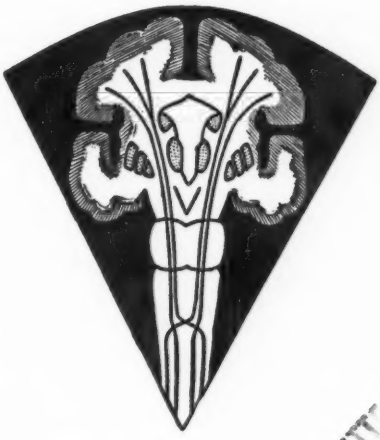

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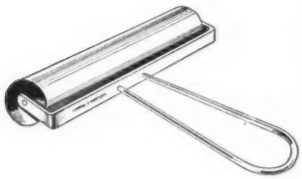

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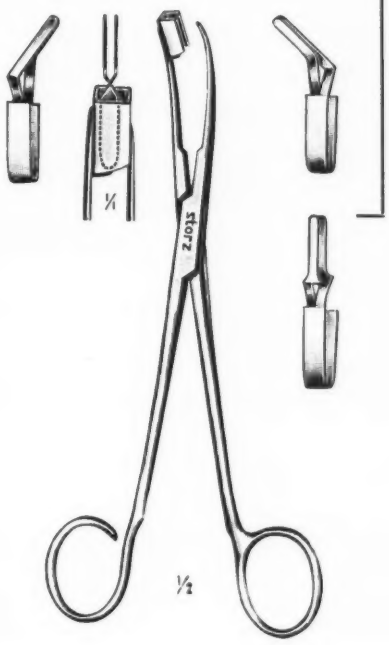

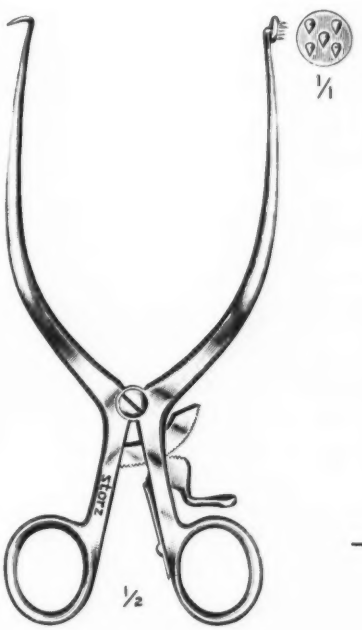

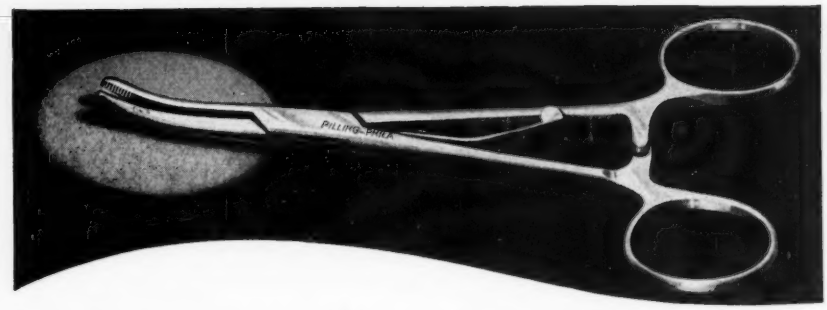

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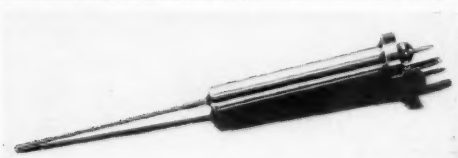

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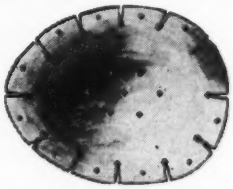

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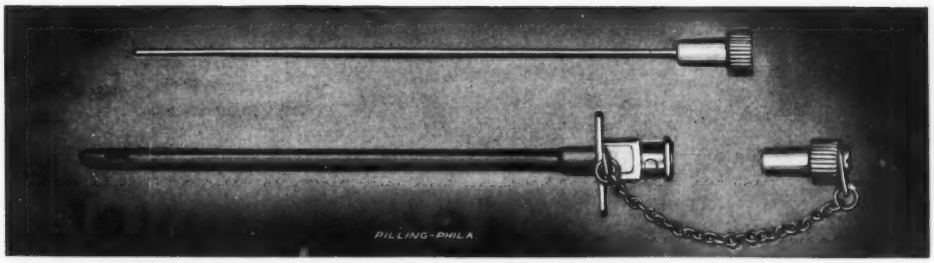

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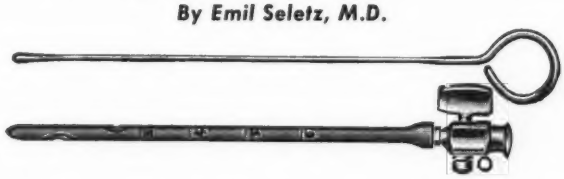

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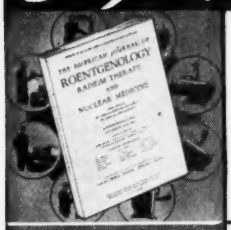

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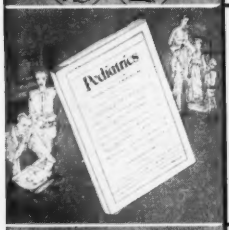

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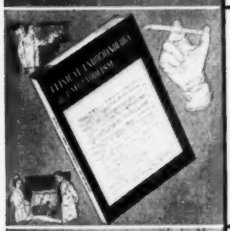

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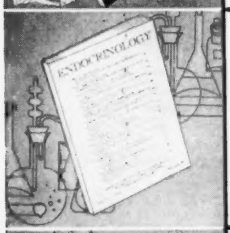

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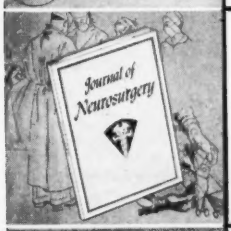

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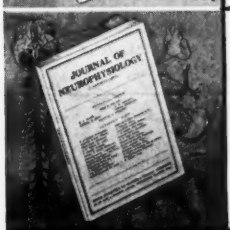

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CEREBRAL PARAGONIMIASIS

REPORT OF FOUR CASES

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(Received for publication October 6, 1954)

PARAGONIMIASIS, or infestation by the lung fluke *Paragonimus westermanii*, is known to be endemic in certain areas of the Far East, particularly in Korea, Japan, and Formosa. In Korea it is found particularly in the areas of Yong Hung, Jun Joo, and the Yak Dong river valley. It is acquired by man by the ingestion of raw fresh water fish, an old traditional delicacy in this part of the world. The disease is so common that it is referred to as "To-Zil," or endemic hemoptysis, and it presents a great problem from the standpoint of medical treatment, public health control and national economy.

It is known that *Paragonimus westermanii* may involve the lungs, pleura, liver, intestinal wall, mesenteric lymph glands, testes, muscles, peritoneum, and brain. The following 4 cases of cerebral cysts caused by paragonimiasis, which were encountered during the last year, are reported because of the rarity of cerebral involvement.

CASE REPORTS

Case 1. An 8-year-old Korean lad was admitted because of motor aphasia, right hemiparesis, and right 7th nerve paresis. He had a history of epileptiform seizures every 2–3 weeks from the age of 3 to 6 years, at which time the seizures stopped but there was insidious onset of right-sided hemiparesis.

Examination. He was a somewhat drowsy boy in no acute distress. His face was slightly puffy. His mentality was low. Positive neurological findings were a gross right hemiparesis, paresis of the right 7th and left 3rd nerves, diminished right-sided stereognosis and sensory response to touch and pin prick, hyperactive deep tendon reflexes on the right side, absent right abdominal and cremasteric reflexes, and a plantar extensor reflex on the right side. Visual fields and optic fundi were within normal limits.

Laboratory tests revealed Hct 36 per cent, Hb. 11.5, RBC 3.82 million and WBC 13,700 with 53 segs., 1 stab., 10 eos., and 36 small lymphs per cent. Urine and CSF were normal. Stool examinations were positive for *Ankylostoma duodenale*, *Ascaris*, *Trichuris trichiura* and *Paragonimus westermanii*. Repeated sputum examinations were negative for *Paragonimus westermanii*.

The chest film was within normal limits. Roentgenograms of the skull revealed nine disseminated calcified lesions scattered in the left frontal and parietotemporal areas, varying in size from a few mm. to 2 cm. (Fig. 1).

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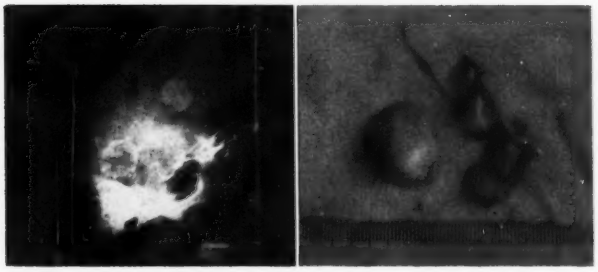
* Chief of Neurosurgery.



FIGS. 1 and 2. *Case 1.* (*Left*) Roentgenogram of skull showing the calcified cysts. (*Right*) The cysts of *Paragonimus westermanii* removed from the brain.

Operation. A left parietotemporal craniotomy was performed and three small calcified cysts were removed from the arachnoid membrane and a 2-cm. calcified cyst was removed from the underlying cortex (Fig. 2). The cysts in the frontal area could not be removed.

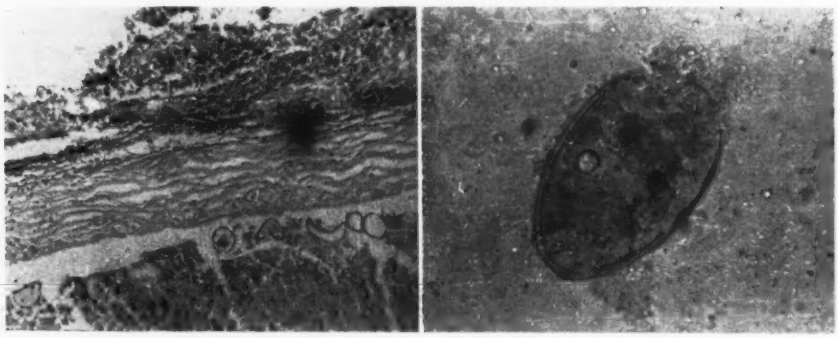
The cysts contained the ova of *Paragonimus westermanii** (Figs. 3 and 4).

Course. Follow-up examination 1 month later revealed marked improvement in the right-sided hemiparesis and sensory defect.

Case 2. A 12-year-old Korean lad was admitted because of nausea, vomiting, headache, and diminished visual acuity. The visual symptoms had been progressive for 10 months prior to admission.

Examination. He was a slightly drowsy boy in no acute distress. The pupils were equally dilated and reacted sluggishly to light. There was bilateral papilledema of 3 D. with a right upper quadrant homonymous hemianopsia and left 3rd nerve paresis. The deep tendon reflexes were bilaterally hyperactive.

Laboratory studies showed a normal hemogram except for 15 per cent eosinophilia. WBC ranged from 6,650 to 15,550. The urine was normal. Stools contained *Ankylostoma duodenale*, *Ascaris*, and *Paragonimus westermanii*. Sputum examinations were negative for *Paragonimus westermanii* and acid-fast bacilli.



FIGS. 3 and 4. *Case 1.* (*Left*) Showing capsule of cyst, its contents with ova, and adjacent damaged brain tissue. (*Right*) The ovum of *Paragonimus westermanii*. The operculated end is becoming destroyed.

* Identified by the United States Army Central Laboratory at Yongdung-Po, Korea.

A chest film was within normal limits. Skull films revealed many calcified smoothly rounded nodules in the left occipital, parietotemporal and frontal areas, varying from about a few mm. to 1 cm. in diameter.

Operation. A left parietotemporal craniotomy was performed. Six superficial calcified cysts were removed from the cortex of the left parietotemporal lobes with moderate damage to the surrounding cortical tissue. The lesions in the frontal and occipital areas were not removed.

The ova of *Paragonimus westermanii* were later identified in the removed cysts.

Course. Following the operation a right hemiplegia and slight verbal aphasia developed.

Case 3. A 22-year-old Korean male was admitted because of left hemiparesis, headache, epileptiform seizures and aphasia. The seizures had been occurring for 10 years, but the other symptoms were of a few months' duration.

Examination. The patient appeared chronically ill with slight pallor and facial puffiness. There was bilateral anosmia. The right pupil measured 4.5 mm. in diameter and the left pupil 3 mm. Pupillary light reflex was absent on the right side only. The corneal reflex was absent on the right side. There was a right 3rd cranial nerve paresis with diplopia. A left homonymous hemianopsia was present. There was no papilledema. Hearing was diminished on the right side. On the left side there were noted hemiparesis, decreased touch and pin-prick sensation, absent abdominal reflexes, hyperactive deep tendon reflexes, ankle clonus, and an extensor plantar reflex.

Laboratory tests revealed a normal hemogram except for 12 per cent eosinophilia, normal urine, and a normal CSF. WBC ranged from 6,300 to 20,300. Stools contained the ova of *Ascaris* and *Paragonimus westermanii.*

A chest film was not remarkable. Skull films revealed many calcified smooth rounded masses of variable size scattered throughout the right frontal, parietal, temporal, and occipital areas.

Operation. A right parietotemporal craniotomy was performed. In the temporal area, 4 cm. above the margin of the helix, a 2-cm. calcified cyst was removed from the subarachnoid space. Immediately below this mass a separate cyst of the same size was found and removed, revealing still a third and deeper calcified cyst, 4 cm. in diameter, which was also removed.

Course. Since operation there has been no significant change in the patient's symptoms or signs except that the epileptiform seizures are less frequent.

Ova of *Paragonimus westermanii* were identified in the cysts removed, but no adult flukes were found.

Case 4. A 32-year-old Korean male was transferred to the neurosurgical service because of aphasia, weakness of the right arm, and clonic seizures starting in the right hand and progressing up the arm to the right side of the face.

One and a half years previously he had had onset of right-sided pleuritic pain. He was admitted to the medical service 10 months prior to transfer. At that time chest films demonstrated probably active apical tuberculosis bilaterally with fluid in the right pleural cavity. He had repeated thoracenteses with removal of small amounts of cloudy yellow or serosanguineous fluid. Cultures of this fluid in our laboratory were negative for acid-fast bacilli as well as for other bacteria. The patient was considered to have tuberculous empyema and active pulmonary tuberculosis though the sputum smears and cultures were also negative for acid-fast ba-

cilli. He was treated with streptomycin 1 gm. twice a week and PASA 12 gm. daily and his pulmonary infiltrations cleared rapidly and the pleural fluid disappeared.

Four months prior to transfer he began to have generalized seizures, which occurred about once a month. None of these seizures was observed. Also at that time he had onset of clonic seizures starting in the right thumb, and then spreading to the right forearm and from there to the right side of the face. These episodes occurred several times a week and were not accompanied by unconsciousness. Some of the generalized seizures apparently were preceded by these localized seizures. About 3 months before surgery there was insidious onset of motor aphasia, weakness and adiadokokinesis of the right arm. Repeated skull films and CSF studies had revealed no abnormalities.

Examination upon transfer revealed evidence of pleural thickening on the right side, slight right 7th nerve paresis, weakness, astereognosis and adiadokokinesis of the right arm, bilaterally hyperactive deep tendon reflexes, more marked on the right side, slight aphasia, diminished sensory perception on the right side of the body, and bilateral Hoffmann sign.

Laboratory studies revealed normal blood serology and urine. Hemogram showed no abnormalities other than a 16 per cent eosinophilia. WBC ranged from 6,000 to 14,000. Stool examinations revealed *Ankylostoma duodenale*, *Ascaris*, *Clonorchis sinensis*, *Trichuris trichiura*, and the ova of *Paragonimus westermanii*. The stools were intermittently guaiac positive.

Repeat skull films showed an area of slightly increased opacity in the left frontoparietal area. Pneumoencephalograms demonstrated the right anterior horn and septum pellucidum to be shifted to the right.

A preoperative diagnosis of left frontoparietal tuberculoma was made.

Operation. A left frontoparietal craniotomy, exposing areas 4, 6, 8, 44 and 45, was performed. A mass composed of 5 small adjacent cysts was removed from areas 8, 44 and 45.

At the time of operation an adult *Paragonimus westermanii* was identified. The fluid in the cysts was thick and yellow. Microscopic examination of the cysts revealed many ova of *Paragonimus westermanii*.

Course. Postoperatively the patient has less aphasia, fewer seizures of the right arm, and greater strength in his right arm. He has had no generalized seizures.

DISCUSSION

Although it is difficult and hazardous to draw conclusions from a series of only 4 patients, certain features are worthy of note.

Three of the 4 patients were infected before they were 12 years of age. One was 32 years old at the time of surgery. In 3 cases epileptiform seizures were an early symptom. One patient (Case 2) entered with symptoms of increased intracranial pressure. All patients with symptoms of more than a year in duration had visual disturbances. In 3 cases there was diplopia with a 3rd nerve paresis. This may have been caused by cysts in the temporoparietal area with pressure of the hippocampal gyrus on the 3rd nerve. All 4 patients had a somewhat lowered intellectual ability, perhaps related to frontal lobe involvement by the cysts. Aphasia was noted to a degree in all patients and perhaps was related to involvement of the speech centers. All patients were right-handed.

The cerebral cortex and subarachnoid space were the main areas of involvement. In all cases there was involvement of the frontal, temporal, and parietal lobes and in Cases 2 and 3 the occipital lobe was also involved. None had apparent cerebellar lesions. All patients had multiple cysts scattered throughout only one hemisphere. In 3 of the 4 cases the left cerebrum was involved; this may be related to the fact that the left common carotid artery is a direct branch of the aorta whereas the right is a bifurcation of the right subclavian artery. The larval forms are thought to spread in the body by blood or lymphatic vessels.[1]

All the cysts were thick-walled and many were calcified. The amount of calcification and the size of the cysts appeared to be related to the duration of symptoms. The perfectly encapsulated cysts were easily removed but several had areas of indistinct boundaries, which made their removal difficult.

Most of the patients had an eosinophilia; however, this was in no way diagnostic as all had many intestinal parasites. The white blood cell count was occasionally elevated.

Ova of *Paragonimus westermanii* were found in the cysts removed in each case. In only 1 case was an adult form found. This was found at the time of operation and placed in saline where it remained apparently viable for 4 hours.

The literature contains references to cerebral paragonimiasis, mainly the Japanese literature. Ko[2] noted 35 cases of cerebral involvement in a series of 95 cases of unusual localization in paragonimiasis, and Matsumura[3] reported 87 cases of cerebral paragonimiasis. Toda[4] reviewed the literature on cerebral paragonimiasis up to 1950 and found 150 cases, in 5 of which surgery was performed. In Korea many studies of paragonimiasis have been made at Seoul University, but cerebral involvement has not been noted.

The symptoms of cerebral paragonimiasis may resemble those of brain tumor, cerebral thrombosis or embolus, meningitis, or Jacksonian epilepsy.

In Korea we are handicapped by having relatively few physicians, particularly neurologists and neurosurgeons. The correct diagnosis, and hence treatment, of diseases of all types is further held back by the prevailing modes and laws in Korea with respect to necropsy.

SUMMARY

Four patients with symptoms of intracranial masses were found at surgery to have intracranial paragonimiasis. The diagnosis in 3 cases was suggested by skull films showing multiple calcified nodules; in 1 case pneumoencephalograms demonstrated the presence of a space-occupying mass. In 3 of the 4 cases it was impossible to remove all the masses visible in the skull films. Three of the patients appeared to have been benefited by surgery.

It is felt that the frequency of intracranial paragonimiasis in the Far East, especially in Korea, is greater than has heretofore been thought. When these cysts produce symptoms of an intracranial mass, they should be re-

moved surgically. There are no good studies of the course of asymptomatic intracranial cysts caused by *Paragonimus westermanii*.

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THE SYNDROME OF ACUTE ANTERIOR SPINAL CORD INJURY*

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IMMEDIATELY following trauma to the spine a syndrome of acute anterior spinal cord injury may sometimes be observed. This is characterized by an immediate complete paralysis with hypesthesia and hypalgesia to the level of the lesion together with the preservation of touch, motion, position and some of vibration sense. This syndrome may be the result of acute anterior spinal cord compression by a dislocated bone fragment or herniated disc or actual destruction of the anterior portion of the cord. Since a surgical lesion causing spinal cord compression is indistinguishable from a nonsurgical destructive one, the author feels that all of these patients should have an exploratory laminectomy and many of them should have a spinal fusion secondarily 2 or 3 weeks later. Emphasis should be placed upon the fact that these patients have attained their maximal neurological deficit and they do not present the usual criteria for surgery, namely, progression of neurological signs or evidence of blockage of the cerebrospinal fluid on the jugular vein compression test.

Thirteen cases are presented in detail to illustrate the pathological conditions producing this neurological pattern and the problems involved in their treatment.

In 1947 Kahn[2] published a report which first described the rôle of the dentate ligaments in chronic anterior spinal cord compression by a posteriorly protruded cervical nucleus pulposus. Spasticity, hyperreflexia, disturbance of gait, weakness, and subjective sensory disturbances suggested the diagnosis of lateral sclerosis. Previously it had been thought that these symptoms were on the basis of compression of the anterior spinal artery, but this paper suggested that the spinal cord usually was firmly fixed over a posteriorly displaced disc by strong dentate ligaments, and that there was exertion of stress just above the dentate ligaments, resulting in pyramidal tract involvement. Kahn stated:

"In anterior spinal cord compression I believe that, with pressure over a period of time, the pyramidal tracts, because of the greater stress on them and the large size of their fibers, have more disturbance of conductivity than the pain fibers of the spinothalamic tracts, even though the latter are closer to the compressing mass, be it midline herniated nucleus pulposus or tumor. Touch is preserved because, even

* A part of the material in this paper was presented in the Symposium on Trauma at the Clinical Congress of the American College of Surgeons, New York City, September 25, 1952.

though the touch fibers of the ventral spinothalamic tracts may fail to conduct, there is still sufficient sensation carried in the more protected posterior columns to prevent the clinical detection of touch disturbance. Postural sense is preserved because the attachment of the dentate ligaments prevents the posterior columns, which are farthest from the compressing mass, from being pressed against the unyielding laminae" (Fig. 1).

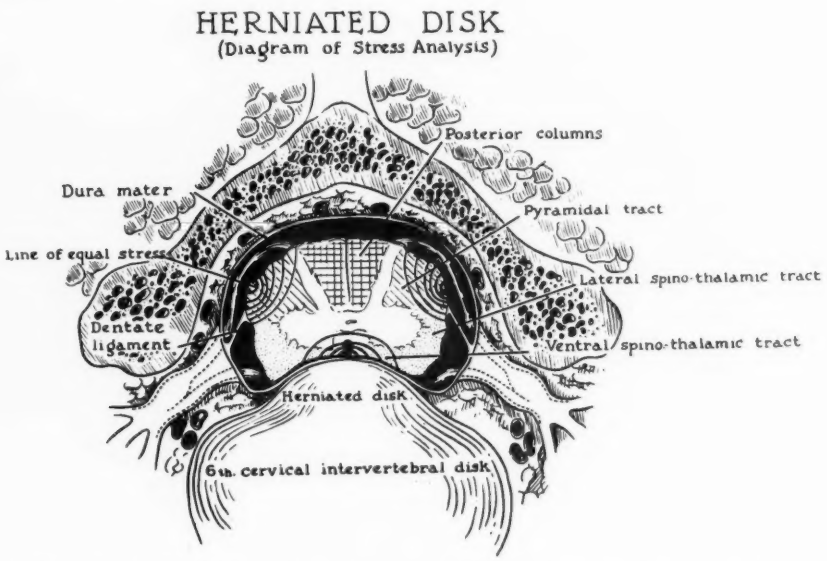


FIG. 1. *Case 1.* "Showing lines of stress in anterior spinal cord compression. Greatest stress is anterior on tracts disturbance of which would not be demonstrable by clinical tests. Secondary stress is directly on pyramidal tracts. The leg area is most lateral in pyramidal tracts, while the hand area is most medial, explaining usual sparing of the hands."[2] (Courtesy of Dr. Edgar A. Kahn)

Bucy *et al.*[1] regarded this mechanism as a possible explanation of the gradual development of neurological symptoms in these chronic cases.

In 1951 the author reported the first 2 cases[3] of what was regarded as an acute phase of Kahn's chronic cervical cord compression mechanism. This topic will be further developed in this paper. The photographs and a brief summary of the original 2 cases are reprinted here to aid in portraying the entire problem.

CASES

Case 1. J.S., a 36-year-old male, was pinned under his truck in an accident on Aug. 21, 1948. At St. Luke's Hospital, Cleveland, he was found to have had immediate complete paralysis with a level of hypesthesia and hypalgesia to the C7 dermatome with preservation of touch. Cervical roentgenograms showed no fracture-dislocation; a lumbar puncture with jugular vein compression test showed no block. After no change in neurological status for 11 days a preoperative diagnosis of pos-

sible herniated cervical nucleus pulposus was made. Laminectomy was performed at C5, C6 and C7. The cord appeared normal. Transdural exploration revealed a totally extruded disc at C6-C7 interspace (Fig. 2). Within 11 days the patient moved his legs and a week later had some return of bladder control.

Two years after injury the patient had a residual Brown-Séquard pattern. He required one cane but worked as a nurseryman and could supervise the care of his sixty-four acres of trees.

FIG. 2. *Case 1.* "The 7th, 6th and 5th cervical nerve roots are exposed, and an extruded nucleus pulposus (arrow) is seen protruding through an incision in the anterior surface of the dura between the 6th and 7th nerve roots."[3] (Courtesy of *Journal of Neurosurgery*)

Case 2. R.C., a 21-year-old college football player, sustained a cervical injury on Nov. 13, 1948. At Crile Veterans Hospital, Parma, Ohio, he was found to have had immediate complete paralysis with hypesthesia and hypalgesia to C6 dermatome bilaterally but touch and part of vibration sense were preserved. On lumbar puncture there was no block on the Queckenstedt test. Cervical roentgenograms showed a severe compression fracture-dislocation of the C5 vertebral body with some displacement of the inferior margin of the body posteriorly into the spinal canal (Fig. 3). It was suggested that the slight posterior displacement of the vertebral body could cause the symptoms, but that a herniated disc at C5-C6 interspace could not be excluded as a possibility. Laminectomy of C4, C5 and C6 was performed. A depressed fracture of C5 lamina was noted bilaterally. The normal-appearing spinal cord was displaced posteriorly. Transdural exploration revealed ex-

truded disc tissue in the spinal canal with several pieces of bone attached to it. There was also a slightly elevated bony ridge.

Three weeks later there was movement in the lower extremities. Twenty-one months after injury the patient drove fifty miles alone. He had some spasticity in his lower extremities but could walk on flat ground without aid. The patient was continuing his physical education career.

As a result of the study of the 2 cases presented in the original article, it was believed that one should add the acute anterior cervical cord injury syndrome to the list of indications for early operation in cases of acute spine injuries. Since that report 11 more cases of this syndrome have been added.

FIG. 3. *Case 2.* "Lateral x-ray showing compression fracture of the body of the 5th cervical vertebra and slight posterior displacement of the body."[3] (Courtesy of *Journal of Neurosurgery*)

Case 3. At 3:45 P.M. on July 8, 1951, J.W., 19-year-old white male, dove into two feet of water, striking the lake bottom and had to be carried out by companions because of the immediate paralysis of all four extremities.

Examination. Upon admission to University Hospital, Ann Arbor, 3 hours later, he had a level of hypesthesia and hypalgesia to the C5 dermatome on the right and C6 on the left. Touch, motion and position senses were intact. Vibration sense was diminished below the level of the lesion. Deep pain was absent below the epigastrium. Supination of both forearms was possible but otherwise there was an areflexic motor tetraplegia. Priapism was present.

Roentgenograms of the cervical spine showed a compression fracture-dislocation of C5 vertebral body with a posterior fragment projecting into the spinal canal (Fig. 4). A lumbar puncture revealed normal spinal fluid dynamics.

Operation. At 9 P.M. on July 8, Crutchfield tongs were inserted and a laminectomy of C4, C5 and C6 was performed. The arch and spinous process of C5 vertebra were found disrupted and lying free. The dura mater was opened in the midline leaving the arachnoid intact, and the cord did not pulsate until the dentate ligaments had been cut bilaterally at two levels. There was no contusion or softening noted on the posterior and lateral aspects of the cord. Palpation and inspection revealed no disc, but only the bony ridge of the posteriorly dislocated C5 vertebra protruding anterior to the cord. The dura mater was left open to prevent any acute spinal cord constriction.

Course. Postoperative roentgenograms on July 24, 1951 demonstrated only partial realignment of the cervical spine (Fig. 5). Unfortunately the Crutchfield tongs

were removed inadvertently on Aug. 7, 1951, and roentgenograms demonstrated a marked redislocation of the cervical spine (Fig. 6). The tongs were reinserted and a more satisfactory realignment was accomplished with traction (Fig. 7). Spinal fusion from C2 to T2 was performed on Sept. 11, 1951, using a tibial graft. Traction was removed Oct. 23, 1951 and a Forrester collar was applied for support.

FIGS. 4–7. *Case 3.* (4) Initial injury. Typical acute flexion or "tear-drop" fracture-dislocation. (5) Partial reduction after traction. (6) Redislocation after inadvertent removal of traction. (7) After re-application of traction.

On discharge the patient had a level to pin-prick at the T3 dermatome. Below this level all other sensory modalities were intact. There was good movement in adductor and fair power in abductor muscles of the shoulders. Flexors of the elbows and pronators and supinators of the forearm showed good strength. With the exception of very minimal voluntary movement of the toes of the right foot, there was no motion in the lower extremities. Deep reflexes of both upper and lower extremities were equal and hyperactive, with a positive pyramidal tract sign in the upper extremity bilaterally, and a positive right extensor plantar reflex.

Approximately a year after his injury the patient was readmitted for relief of spasms in his lower extremities. Marked contractures were present in the joints of all extremities. There was moderately good voluntary dorsiflexion of the right foot and toes and some flexion of the right knee, but poor extension of the leg. Little or no voluntary movement was found in the left lower extremity. Marked adduction spasm was present, with intermittent flexion spasms of the left lower extremity. There had actually been only a very slight degree of recovery, for superficial sensation had been intact at the time of discharge from the hospital. The patient refused anterior rhizotomy for spasms in his right leg and was discharged.

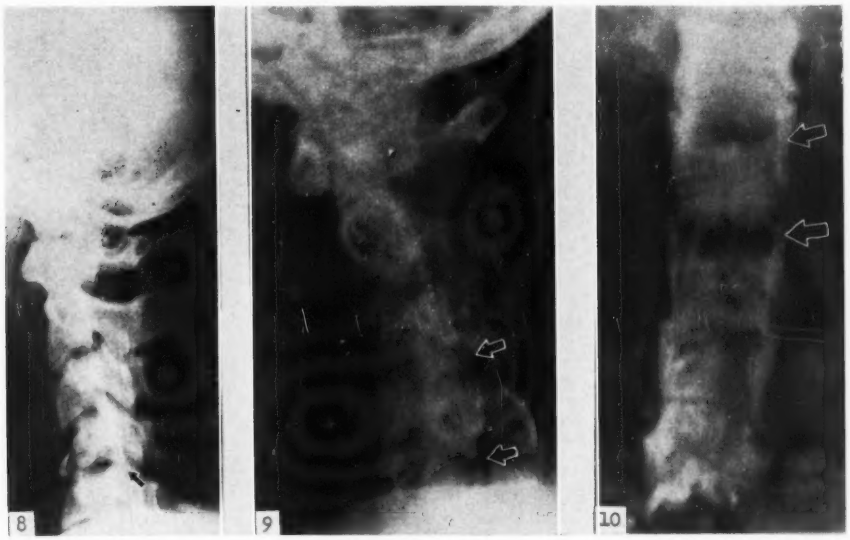
Comment. This patient had the typical acute anterior cervical cord injury syndrome with no evidence of a block on manometric testing of the cerebrospinal fluid. Early operation confirmed the presence of a posterior bony ridge pressing on the anterior surface of the cord at C5–C6 interspace, with no evidence of contusion or laceration. Postoperatively there was some degree of recovery but it was minimal. The neurological residual could have resulted from the severe initial contusion of the cord with irreparable damage or it could have been the result of further injury by redislocation of the spine, or perhaps have been related to the final angulated fusion of the involved vertebra, so that there had been an incomplete correction of the deformity, causing signs of chronic anterior cord compression. A typical "tear-drop" fracture of the C5 vertebral body was noted with the anterior inferior border of the vertebra dropping forward and the posterior inferior border protruding posteriorly into the spinal canal. This was similar to the roentgenographic findings in Case 2.

Case 4. H.P., 16-year-old school boy, sustained a severe cervical spine injury while tumbling in a school gymnasium at 1 P.M. on May 14, 1948. He stated that he had suffered an immediate complete paralysis of all four extremities with a loss of sensation below the neck. A local physician took cervical roentgenograms which revealed a fracture-dislocation of C4 on C5 vertebral body (Fig. 8). He was transferred to University Hospital, arriving at 2 A.M. on May 15, 1948.

Examination. There were minimal movements of the biceps and deltoid muscles with involuntary twitching of the left thumb. No movement was observed in the pectoralis, triceps, or muscles of the forearms or lower extremities, and there was complete areflexia. A lumbar puncture demonstrated a spinal fluid pressure of 150 mm. of water which rose rapidly to 250 mm. upon jugular vein compression bilaterally and then fell slowly, suggesting an incomplete block. The latter was ascribed by the resident to "a faulty mechanism."

2nd Admission, May 1952. The patient's presenting complaint was severe involuntary spasms with contractures of the fingers.

Examination. There was a level at T6 dermatome below which superficial pain and temperature sensations were absent. Hypesthesia was present to C6 dermatome. Motion and position senses were preserved throughout the body. Vibration sense was present except over the right external malleolus. Touch was good except in the left lower extremity where responses were only spottily correct. Generalized spasm was found in all extremities, but the fingers were contracted down into fists and the legs were frequently extended rigidly. There was sufficient spasm occasionally to precipitate the patient out of the bed onto the floor. There was bilateral wrist and left patellar clonus. Reflexes were hyperactive throughout. Extensor plantar reflexes could not be elicited, probably because of extreme extensor spasm.



FIGS. 8–10. *Case 4.* (8) Compression fracture of C4 vertebral body with minimal posterior dislocation of C4 on C5. (9) Four years later. Appearance giving false impression of fusion across interspace with some residual dislocation. (10) Myelogram demonstrating midline defects at C3-C4 and C4–C5 interspaces.

Cervical roentgenograms at this time demonstrated a loss of the intervertebral space at C3-C4, and C4-C5 with apparent fusion and slight anterior dislocation of C3 on C4 vertebral body and posterior displacement of C4 on C5 body (Fig. 9). Lumbar puncture showed a pressure of 150 mm. of water with a prompt rise to 300 mm. on jugular compression and a rapid fall to the original level within 10 seconds. Cervical myelogram (Fig. 10) revealed midline defects in the contrast medium at C3-C4 and C4-C5. Motion, position and vibration senses were further impaired in the lower extremities after myelography.

Operation, May 19, 1952. Cervical laminectomy of C3 through C6, in spite of the apparent fusion on the roentgenogram, revealed marked instability of both the 3rd and 4th cervical vertebra but no evidence of a fracture. There were marked adhesions between the arachnoid and the overlying dura mater laterally at the C4-C5 interspace and after cutting the dentate ligaments at four levels bilaterally, gentle

retraction of the cord showed the bony kyphosis demonstrated in the roentgenogram. No herniated disc could be demonstrated. There was a definite variation in the level of insertion of the dentate ligaments with a more posterior displacement of the spinal cord at C3-C4 and C4-C5 than at the other two exposed interspaces. The dura mater was closed.

Course. Postoperatively the patient lost the wrist clonus, and tonus in the upper extremities was decreased about 50 per cent, but the mass reflexes in the lower extremities were slightly accentuated. Finally anterior rhizotomy was performed in the latter part of June 1952. This procedure adequately controlled the spasms of the lower extremities. Marked spasticity and clonus remained in both upper extremities.

Comment. When this patient was admitted to the hospital at the time of his injury, he had an acute anterior cervical cord injury syndrome. There was no progression of signs and only partial block on jugular compression test, consequently he was not operated upon. No regression of symptoms had occurred over a period of 4 years. The cervical exploration should have been performed as soon after injury as the patient's condition permitted. The pronounced instability of C3 and C4 vertebral bodies even 4 years after injury suggests the importance of early fusion of cervical spines associated with these incomplete lesions, and demonstrates how misleading the roentgenograms may be in interpreting stability of the cervical spine. This is a case of the acute anterior cervical cord injury syndrome without a complete block, which did not show recovery after 4 years. Another "tear-drop" fracture is demonstrated with associated prolonged spinal instability.

Case 5. M.S., a 17-year-old tumbler, slipped and struck upon the vertex of his head at 8:45 A.M. on March 29, 1951. He was not rendered unconscious, but had complete paralysis of all four extremities except for questionable movement of his right great toe. There was headache, with pain in the neck and numbness from the neck downward over the trunk and extremities.

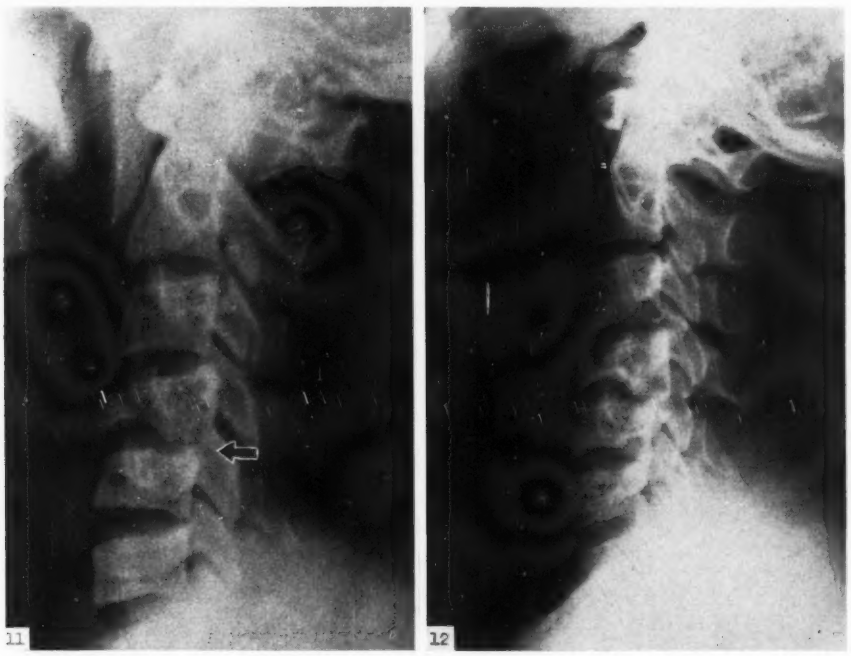
Examination. There were hypesthesia and hypalgesia to the level of the C4 dermatome, but touch and position senses were preserved bilaterally throughout the entire body. Both lower extremities were completely paralyzed. The patient could extend the wrists but had no motion in the fingers. There were no extensor plantar reflexes. Cervical spine roentgenograms revealed a compression fracture-dislocation of the C4 vertebral body with a 3 to 4 mm. posterior displacement (Fig. 11).

Course. Crutchfield tongs were applied. On March 31, 1951, lumbar puncture was done and on jugular compression test there was a rise in pressure to 300 mm. of water and a slow drop suggesting the possibility of partial subarachnoid block. In this spinal fluid specimen with only 1 blood cell/high power field the Pandy test was reported as ± 1 and total protein was 63 mg./100 cc. On April 14, 1951, lumbar puncture showed a rise of pressure to 240 mm. of water and a slow fall to normal. This specimen contained no blood cells and total protein was 110 mg./100 cc.

On April 23, 1951, the sensory level was gradually receding, and there was onset of return of function in the lower extremities with an ability to flex the fingers of both hands. A plaster body jacket was applied on April 27, 1951 at the time of removal of the Crutchfield tongs. A back brace was substituted for this appliance on June 14, and the patient was able to ambulate moderately well. There was a residual urinary volume of 75 cc. On August 3, the patient was up in a walker. It was dis-

covered that his sensory level, which had receded to T12-L1, had now risen again to the T8 dermatome. There was weakness in the extensors of the left wrist with wrist drop. Right foot drop was also present. Bilateral extensor plantar reflexes and ankle clonus were elicited. Bladder control and bowel function were good.

Recheck stability roentgenograms of the cervical spine on May 21, 1952 exhibited motion at C3-C4, while there was none apparent at C4-C5 and at C5-C6 (Fig. 12). By August, 1953 the patient began to have some increasing spasticity in



FIGS. 11–12. *Case 5.* (11) Initial "tear-drop" compression fracture with minimal posterior dislocation of C4 vertebral body protruding into the spinal canal. (12) Healed lesion 2½ years after injury showing bone projecting into the spinal canal.

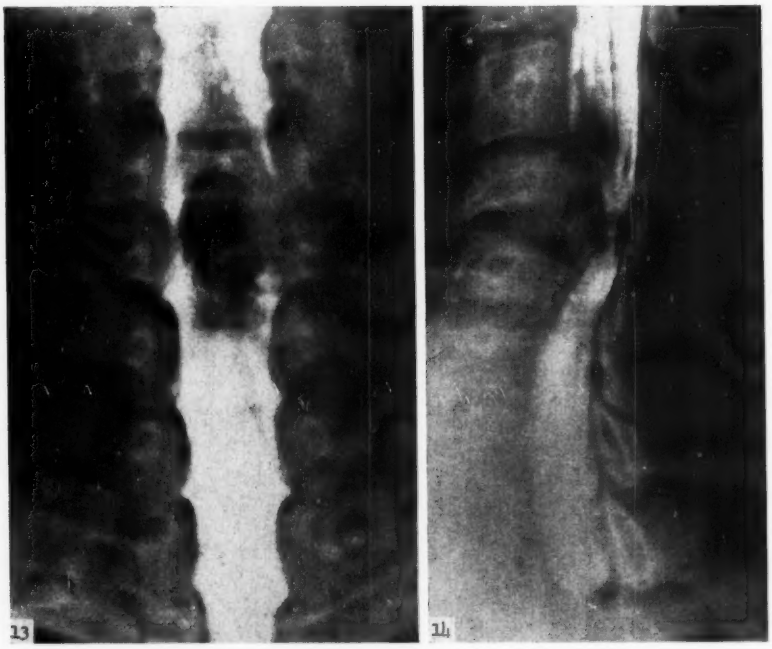
the lower extremities and was readmitted for further studies. A cervical myelogram on Sept. 1, 1953 demonstrated a marked narrowing of the cervical spinal canal (Figs. 13 and 14).

Operation, Sept. 2, 1953. At decompressive laminectomy, bilateral healed fractured laminae of C4 and C5 were removed. These laminae were thinned and osteoporotic. Parts of the C3 laminae were removed. The bony spine was extremely unstable. The dura mater was opened in the midline and the dentate ligaments, which were very thin, were sectioned at two levels. At the C4-C5 interspace there was a 3–4 mm. central bony protrusion. The dura mater was left open, protected by the intact arachnoid in the center of the dural aperture. An inverted V-shaped bone graft was inserted bilaterally from C3 to C6 spinous processes.

Course. Postoperatively the patient was able to walk with a cane, using a spring

kick-up brace. Hypesthesia and hypalgesia were spotty to a level of T10 dermatome on the right side. Temperature sense was not clearly defined over the lower extremities, particularly on the right side. Motion and position senses were intact bilaterally. There were bilateral pyramidal tract signs with increased tonus in the upper and lower extremities, ankle clonus and extensor plantar reflexes bilaterally.

The patient has continued to improve, so that he is now back to his preoperative status.



FIGS. 13–14. *Case 5.* Myelographic defect on anteroposterior and lateral views indicating marked narrowing of cervical spinal canal.

Comment. This patient had a typical acute anterior cervical cord injury syndrome, combined with the frequently observed acute flexion or "teardrop" vertebral body fracture. *It is particularly interesting to note that he did not have a complete block on jugular compression test on two days, 5 days apart, and yet he had a definitely progressively elevated protein without any blood being present in the specimen.* This would suggest irritation of the cord, possibly caused by bony pressure upon its edematous anterior surface, and shows that one does not need a complete block or hemorrhage to have an elevated protein. Perhaps it merely indicates contusion of the cord, but in that case the protein should not have become progressively elevated. Cervical myelography may be extremely dangerous because of the degree of narrowing of the canal. The patient apparently seemed to be making a very nice recovery

from his injury initially but then began to have regression with increasing spasticity. In retrospect, the patient should have been operated upon as soon after his injury as his condition permitted and then either primarily or secondarily had a spinal fusion.

Case 6. L.C., an 18-year-old boy, dove into shallow water at 4 P.M. on July 11, 1953. He sustained immediate paralysis of both legs with marked weakness of his arms and hands, and had to be dragged from the water by his companions. On admission to the hospital the patient complained of pain in the neck.

Examination. There was marked weakness of all movements of the hand, particularly extension and flexion of the wrist. There was complete paralysis of the lower extremities. Hypalgesia and hypesthesia were demonstrated to the level of C7 dermatome. Position sense and motion sense were impaired but not absent, while vibration sense was lost below the level of C7 dermatome. The biceps and triceps jerks were hypoactive, abdominal reflexes were absent, and the knee and ankle jerks were absent. There were no definite extensor plantar reflexes.

Roentgenograms of the cervical spine revealed a fracture of the anterior inferior lip of the 6th cervical vertebra (Fig. 15). Lumbar puncture demonstrated a partial block upon jugular vein compression.

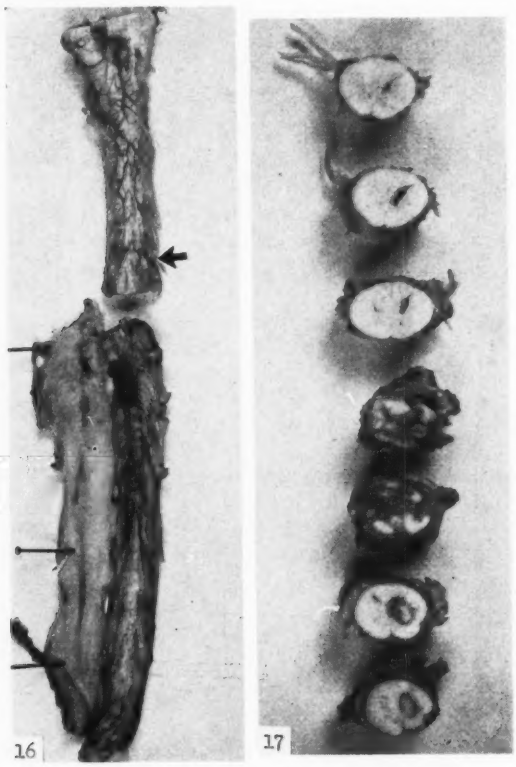
FIG. 15. *Case 6.* Typical flexion fracture-dislocation with C6 vertebral body protruding into the anterior portion of the spinal canal.

Operation. The laminae of C6 and C7 vertebrae were found to be fractured bilaterally and a central fragment was depressed downward on the cord. Decompression was carried out by sharp dissection. The dura mater was opened in the midline, and the cord appeared normal. Upon sectioning the dentate ligaments, two on the right side, and one on the left, the cord pulsated normally. Transdural exploration anteriorly revealed a bony elevation at the C6-C7 intervertebral space. The dura mater was closed tightly.

Course. Postoperatively the patient had some difficulty with atelectasis and mental confusion. On the 4th day after operation while secretions were being removed by suction from the nasopharynx, he suddenly ceased breathing. All attempts to revive him were of no avail.

Necropsy. There was evidence of postoperative extradural hemorrhage at the site of the wound, but the dura mater had been closed tightly. Upon opening the dura mater the cord now appeared to be swollen and purple on its posterior surface

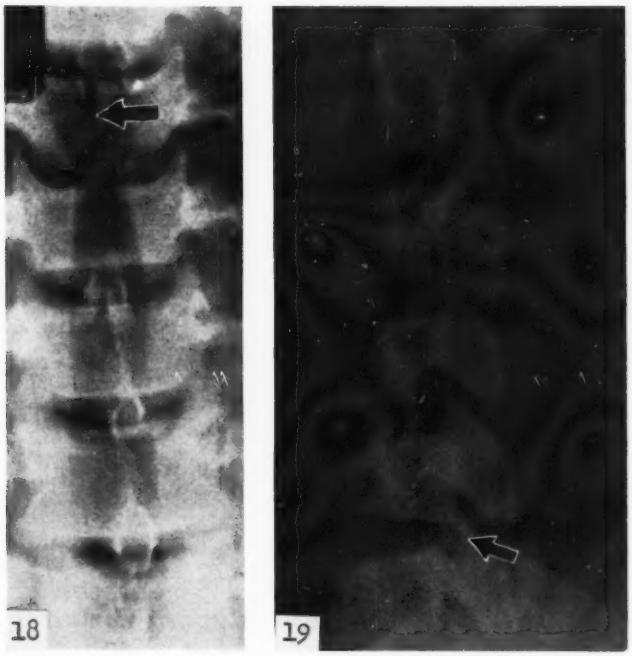
and at the upper margin of the dural incision at C4 lamina there was a definite constriction (Fig. 16). Multiple incisions were made across the cord at the levels of C3 through T2 (Fig. 17). There was definite dissection or cavitation of the lesion as high as the C3 level. At the site of the fracture-dislocation at C5-C6 there was marked destruction of the cord and it is scarcely conceivable that the patient could have had a normal-appearing posterior cord at the time of operation. The damage which involved the central portion of the cord could be visualized to T2 level.



FIGS. 16–17. *Case 6.* (16) Specimen of cervical cord at necropsy 4 days later. Arrow points to cephalad pressure cone at point where C4 lamina indented the cervical cord. Note appearance of the posterior portion of the cervical cord immediately under the site of the dural incision. (17) Transverse section of cervical cord demonstrating the massive damage from C2 to T1 cord segment.

Comment. This patient presented the anterior cord injury syndrome, and is the only one who succumbed to *the spinal cord injury* following operation. There were several errors. First, the extent of laminectomy was not sufficiently large. From this experience the author is inclined to feel that at least three laminae should be removed so that the dura mater may be incised in the midline and the dentate ligaments cut at least at three levels. If this

degree of exposure is not obtained, there is too much of a tendency to press upon the cord in an attempt to reach the dentate ligaments and section them. The orthopedic surgeons are not anxious to have the neurosurgeon remove this amount of bone, but adequate exposure is necessary. The second error was the failure to obtain a completely dry wound and prevent any degree of extradural hemorrhage. Thirdly, it is doubtful whether the dura mater should have been closed over the cord even though the latter structure



FIGS. 18–19. *Case 7.* (18) Original splitting fracture of C5 vertebral body. (19) Lateral view demonstrating compression and minimal fracture dislocation.

appeared to be quite normal and free of edema. One should probably have allowed for more postoperative swelling. There was definite evidence of a constriction at the upper margin of the incision at the C4 level. It was probably a combination of compression by the hemorrhage and the constricting dura mater which accentuated anoxia to the already edematous cord and was responsible for progressive edema to the C4 segment, resulting in a respiratory death. A typical "tear-drop" or flexion fracture was present.

Case 7. C.A., a 14-year-old boy, dove into a shallow pool striking his head on the bottom. He could not move his four extremities upon his removal from the pool. At the local hospital the intern confirmed the fact that there was an areflexic paraplegia,

but merely made the note "sensory examination grossly normal." Priapism developed shortly after the injury. Cervical roentgenograms demonstrated a compression fracture of the C5 vertebra with minimal fracture-dislocation. Traction was applied. Examination 48 hours after admission noted no change in motor power, but a level of hypalgesia above the nipple line with motion and position sense preserved.

On April 16, 1952, 5½ weeks after injury, he was transferred to University Hospital for intensive physiotherapy.

Examination. Vibration, position and touch sensations were intact throughout but there was a definite level of hypalgesia to the T2-T3 dermatome. There was inability to flex the fingers bilaterally with extension occurring at the wrist on attempted flexion. Impairment of extension of the arms at the elbows was present bilaterally with complete paralysis of the lower extremities. Deep reflexes were symmetrically hyperactive with bilateral extensor plantar reflexes and sustained ankle clonus.

Recheck cervical roentgenograms showed a vertical splitting fracture of the C5 vertebral body (Fig. 18) with minimal anterior dislocation of C5 vertebral body on C6 (Fig. 19), suggesting that a bony ridge was pressing anteriorly on the spinal cord. On lumbar puncture the spinal fluid manometrics were normal. A diagnosis of anterior cord compression was made.

Operation, April 18, 1952. A C5, C6 and C7 laminectomy was performed, the dentate ligaments being cut at these interspaces, and thereafter the normal-appearing cord was noted to pulsate freely. No protruded disc could be palpated but the ridge of the slightly displaced C5 vertebral body was noted. The wound was closed in layers.

Course. Within 36 hours the patient had movement of his toes bilaterally and some flexion of the fingers of his right hand. By April 27, 1952 there was some improvement in strength in the lower extremities and marked return of motor function in the upper extremities, including the intrinsic hand muscles. His indwelling catheter could be removed. Because of a tendency toward moderate kyphosis (Fig. 20) on removal of the traction apparatus 9 weeks after injury a spinal fusion was performed on June 2, 1952 (Fig. 21).

Comment. This patient had the typical anterior cervical cord injury syndrome. Since there was no block on manometric studies, critics will observe that this patient might have recovered spontaneously. However, after 5½ weeks with no improvement in neurologic status, there was a recovery of motor power in the fingers and the toes within 36 hours postoperatively. This quick response to decompression appears to be more than a mere coincidence. The minimal degree of dislocation of the anterior floor of the spinal canal was probably sufficient to cause pressure on the anterior cervical spinal cord, which was restrained by the dentate ligaments. The force of the injury was not severe enough to pinch off the anterior inferior margin of the vertebral body to form the typical flexion fracture.

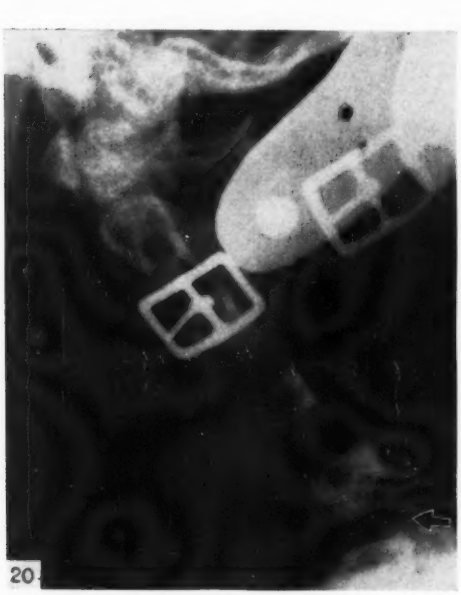
Case 8. S.S., a 31-year-old man, was involved in an automobile accident at 1:30 A.M. on June 29, 1952. He was immediately paralyzed.

Examination. Upon admission to the University Hospital at 6 A.M. a 1 cm. right frontal scalp laceration was noted. There was a complete flaccid paralysis of the lower extremities with some weakness of the biceps muscles bilaterally. There was

a level of hypalgesia and hypesthesia to C7 dermatome bilaterally. Touch, motion and position senses were intact. Areflexia of the lower extremities was present with an absence of the right biceps reflex. The biceps jerk on the left was present and there were bilateral hypoactive triceps reflexes.

Cervical spine roentgenograms revealed a fracture-dislocation of C6 vertebral body anteriorly on C7 with comminution of the C7 vertebral body (Fig. 22). There was displacement posteriorly of the superior rim of the C7 vertebral body into the spinal canal.

1st Operation. The right frontal scalp laceration was debrided by Dr. Lloyd Lemmen at 8:30 A.M. and a linear fracture was demonstrated which extended into



FIGS. 20–21. *Case 7.* (20) Tendency toward recurrent dislocation after removal of Crutchfield tongs. (21) After spinal fusion.

the right temporal area. Two burr holes over the site of the fracture showed a small extradural hematoma. Crutchfield tongs were applied.

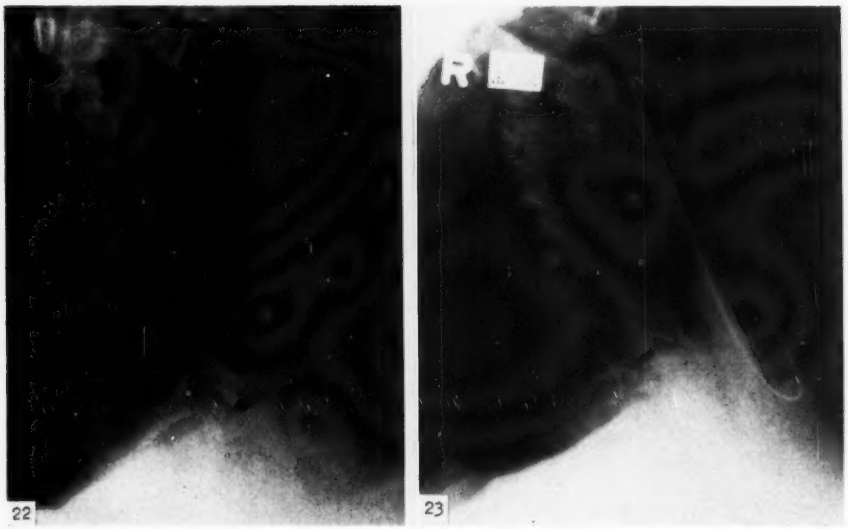
A laminectomy of part of C4, C5, C6 and part of C7 laminae was performed. It was found that the laminae of C5 and C6 were fractured bilaterally and were impinging upon the dura mater. There was a moderate amount of extradural hemorrhage. The dura mater was opened in the midline, and the spinal cord appeared normal. The dentate ligaments were cut at three levels. The dura mater at the point of exit of the C6 nerve root was torn and had to be sutured. The remainder of the dura mater in the midline could be readily closed.

Course. Because of respiratory distress, a tracheotomy was performed at 12:30

P.M. The patient's arms showed an increase in strength shortly after operation. On July 11, 1952 he exhibited the first voluntary movement in his lower extremities.

2nd Operation. On Aug. 6, 1952, a cervical spinal fusion was performed, using a left tibial bone graft (Fig. 23). This was fitted in place as a clothes-pin graft between the spinous processes of C2 and C7 and was fastened with #16 steel wire.

Course. On examination on Aug. 10, 1952 there were good movements in all four extremities, including the toes. There was a vague level of hypalgesia to the C6 dermatome, but touch, motion, and position senses were intact. Sensation was now noted on irrigation of the bladder, but true bladder control had not been established.



FIGS. 22–23. *Case 8.* (22) Initial injury showing bone fragment projecting into anterior portion of canal (23) Realignment by skeletal traction for a period of 5 weeks followed by tibial graft fusion.

On follow-up examination on July 15, 1954 good motor power with increased tone was elicited in all four extremities and there was almost complete recovery of sensation. Bladder control still was poor, requiring the use of a leg urinal during working hours. He is now working full time selling electrical appliances.

Comment. This case presented a typical pattern of an anterior cervical spinal cord compression. The tracheotomy was necessary to remove tracheobronchial secretions adequately. Surgical intervention seemed to be of definite value to this patient.

Case 9. K.L., a 41-year-old farmer, while driving his truck ran off the road and hit a tree on Dec. 29, 1952. There was an immediate paralysis of all four extremities, except for minimal movement in the fingers of the right hand. He was numb from the chest down but could perceive touch. There was no interval of unconsciousness. About the 2nd day the patient began to have some return of movement in his arms, but the legs remained paralyzed. There was no bladder and bowel control, but the

patient could distinguish bladder distention during irrigation. He was having some respiratory difficulty and exploration at this time was deemed inadvisable.

Examination revealed about 25 per cent impairment of the biceps muscle function bilaterally. There was complete flaccid paralysis below this level. Light touch, deep touch, and position senses were preserved. Partial vibration sense was noted in the ankles and at the knees. There was loss of pain and temperature sensations to level of T4. Biceps reflexes were faint bilaterally; triceps reflexes were absent. There was a complete lower extremity areflexia.

Cervical roentgenograms showed an acute flexion fracture of C6 vertebral body with dropping forward of anterior inferior margin and posterior margin backward into the spinal canal (Fig. 24). Lumbar puncture revealed clear colorless CSF. Queckenstedt test was negative.

FIG. 24. *Case 9.* Fracture-dislocation with minimal displacement of posterior inferior portion of the C6 vertebral body into the anterior portion of the spinal canal.

Operation, Jan. 15, 1953. Laminectomy was done with removal of the laminae of C4, C5 and C6. All of these laminae were fractured, but not depressed. The dura mater was intact, and upon opening it in the midline the spinal cord appeared to be normal. Exploration anterior to the cord showed no evidence of any disc or anterior spinal cord compression. The dura mater was closed in the midline since the cord did not appear swollen.

Course. Six months postoperatively the patient had slight movement in the toes of the right foot and was able to flex the right leg slightly at the knee. He was able to close the fingers of the right hand fairly well, but the grip of the left hand was markedly impaired.

Comment. This patient was not operated upon early because of rather marked respiratory embarrassment at the time of admission. There was some posterior displacement of the body of the C6 vertebra into the spinal canal. The typical "tear-drop" fracture of the anterior portion of the body was found. Unfortunately, this patient probably had more anterior cord destruction than compression.

Case 10. M.V., a 40-year-old white female, was riding in a truck with her husband when it overturned at 3 A.M. on May 8, 1953. She was not rendered unconscious and noted immediate complete paralysis with numbness and tingling in her

arms and legs. She was admitted to University Hospital a few hours after her injury.

Examination. There was no movement of the arms or legs. Position, deep pain, and touch sensations were present. Vibration sense was impaired over the malleoli and iliac crest bilaterally. Hypalgesia and thermanesthesia were present to the level of C6 dermatome. There were pyramidal tract signs in the upper extremities and there was withdrawal on attempting to elicit extensor plantar reflexes. The biceps, triceps, knee, and ankle jerks were all mildly hypoactive. Upon lumbar puncture no block was found on performing the Queckenstedt test. The CSF was bloody.

Roentgenograms of the cervical spine revealed a unilateral dislocation of C3 on C4 vertebra with disruption of the unco-vertebral joint on the right side (Fig. 25).

FIGS. 25–26. *Case 10.* (25) A unilateral dislocation of C3 on C4 cervical vertebra with disruption of the unco-vertebral joint on the right side. (26) Reduction of fracture-dislocation and fusion using steel wire with an overlying roof of iliac bone graft.

Course. On May 16, 1953 the patient recovered the first faint movements of the toes on both feet. She was transferred to her local hospital for a period of 5 weeks and then readmitted to University Hospital.

She had improvement in flexion of the hands with greater power on the left side. There was fair strength in both triceps muscles. Movement was now present in all muscle groups in the lower extremities, being more marked on the left side. No definite sensory deficit could be established. There was hyperactivity particularly of the lower extremity reflexes, with bilateral ankle clonus. There were bilateral extensor plantar reflexes.

Check cervical spine roentgenograms demonstrated a very minimal dislocation at the C3-C4 intervertebral space.

Operation. Complete laminectomy was done of C3 and C4 vertebrae and the dura mater was opened in the midline, preserving the arachnoid. The arachnoid was then incised laterally so that the dentate ligaments could be cut at two levels. Palpation anterior to the cord demonstrated a slight dorsal protuberance at the C3-C4 in-

tervertebral space. The dura mater was closed in the midline, and a firm iliac bone graft was inserted between the spinous processes of C2 and C5. This graft consisted of an imbricated notched single piece of bone inserted between the spinous processes on either side. Bone fragments were then placed along the margins of the roughened facets at the lateral border of the wound. A piece of wire was used to maintain position of the graft, being anchored to the C2 and C5 spinous processes (Fig. 26).

Course. Follow-up examination on June 28, 1954 showed that the patient had some spasticity in all four extremities but she could walk with crutches. She was able to use her hands well enough to carry out 60 per cent of all functional tests. With more intensive physiotherapy she should have an excellent prognosis.

Comment. It would have been dangerous to operate upon this patient at the time of admission in view of the possibility of producing phrenic paralysis because of the high site of the lesion. In retrospect it would have been much better to have applied traction, reduced the fracture-dislocation with tongs, and performed a laminectomy with section of dentate ligaments 2 or 3 weeks after injury. Spinal fusion could probably have been performed at the same time as the laminectomy, thereby saving 5 or 6 additional weeks in skeletal traction.

Case 11. R.S., a 19-year-old male, was involved in an automobile accident on Aug. 20, 1950. He was unconscious for 15 minutes and had immediate paralysis of arms and legs, but it was reported that "some sensation was intact." Motor power was never regained in any of his extremities. The patient was placed on a Stryker frame in October 1950, and for a period of 3 months prior to transfer he had marked flexion spasms of the upper and lower extremities. On March 2, 1951 he was admitted to University Hospital.

FIG. 27. *Case 11.* Lateral roentgenogram showing minimal amount of displacement at C4–C5 interspace.

Examination. There was a good range of motion present in the neck, but there was spastic paralysis of all four extremities with slightly more active movement in the right shoulder than the left. Touch sense was preserved over the entire body. Motion and position senses were absent in lower extremities but present in the upper. Impairment of vibration sense was noted to the T7 dermatome level. Hypalgesia was present to C4 dermatome. There were normal knee and ankle jerks but the biceps and triceps were symmetrically hyperactive. Mass reflexes occurred bilaterally upon the slightest stimulation. A definite left extensor plantar reflex was observed with an equivocal one on the right.

Cervical spine roentgenograms revealed a compression fracture of the superior

aspect of C5 with minimal anterior dislocation of the C4 vertebral body on C5 (Fig. 27). Lumbar puncture revealed no block on manometric study and a total protein of 31 mg./100 cc.

A diagnosis was made of a posteriorly protruded disc or pressure on the anterior surface of the cord because of the bony ridge of the dislocated vertebral body.

Operation, March 6, 1951. Laminectomy was performed at C4, C5 and C6. Marked instability of C5 and C6 vertebrae was demonstrated. Little epidural fat was found at this level and the ligamentum flavum at the C4-C5 interspace was torn. The dura mater was opened in the midline. Adhesions bound the arachnoid to the spinal cord posteriorly at the posterior columns. The dentate ligaments were sectioned at three levels to permit good exposure and anterior exploration revealed a slight bony posterior projection of the C5 vertebral body. Firm adhesions had to be dissected away gently anteriorly and at this point in the procedure the patient ceased breathing on several occasions, but respirations started after manipulation of the cord was halted. No protruded disc was found. At the level of the adhesions there was also firm scarring of the cord. The dura mater was closed, and the wound was sutured in layers.

Course. The patient was discharged on April 22, 1951 with no change in his neurological status except that there was some adduction and abduction of the right thumb, but unfortunately his spasms were slightly more severe. Anterior rhizotomy was rejected by the patient and relatives.

Comment. This patient was seen 7 months after his injury and presented the picture of anterior cervical cord injury without a block on spinal fluid manometric studies. There could have been an immediate complete tetraplegia with a blockage of the spinal fluid on jugular compression test immediately after injury. The symptoms might, on the other hand, have been caused by prolonged spinal cord edema which left the patient with the syndrome of anterior cord compression, or possibly by traumatic thrombosis of the anterior spinal artery. The latter, however, may cause segmental changes. If there had been a spinal fluid block immediately after injury, according to the usual criteria the patient should have been operated upon. We feel that even if there was no obstruction on the Queckenstedt test the neurologic pattern of anterior cervical cord injury would have suggested the possibility of a surgical lesion requiring decompression. Operation was performed in this case with little hope of accomplishing anything. It was urgently requested by the patient and his family.

Case 12. A.C., a 53-year-old man, was involved in an automobile accident on March 14, 1951. He was able to walk away from the site of the accident, but about 5 minutes later there was incomplete paralysis of his arms and legs. He was taken to a local hospital, and 5 days later, when his condition became progressively worse, he was transferred to University Hospital, on March 19, 1951, at 6 P.M.

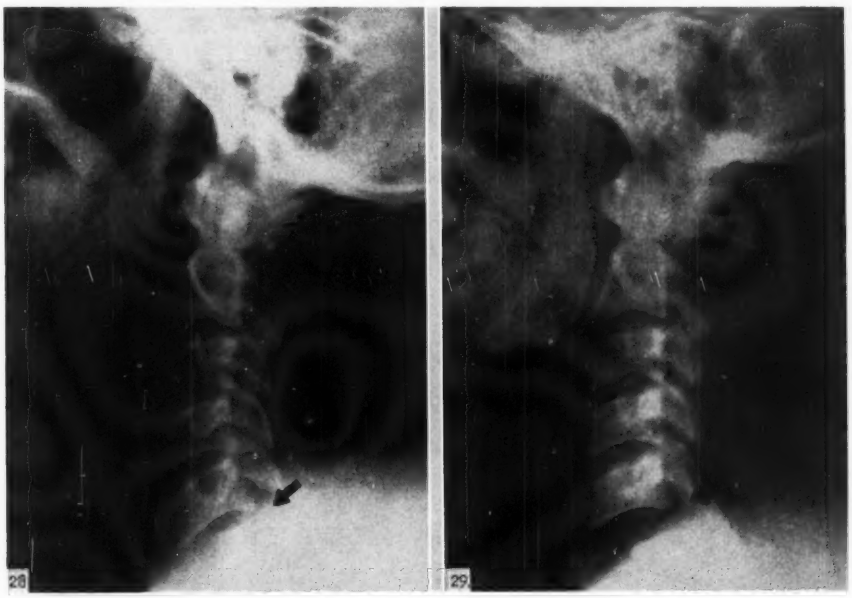
Examination. The patient was able to move his toes very slightly, but otherwise there was little movement in the lower extremities. At 7:30 P.M., it was observed that there was complete paralysis of the lower extremities with no movement of his fingers and there was weakness of the triceps and biceps, bilaterally. Motion, position, vibration and superficial touch sensations were intact. There was a vague level of hypalgesia below T9 bilaterally. Deep reflexes were equal and active bilaterally,

and there was an equivocal extensor plantar reflex bilaterally. The patient was incontinent of urine and his abdomen was markedly distended.

Cervical roentgenograms revealed a fracture-dislocation of C5 vertebral body on C6 (Fig. 28). Crutchfield tongs were inserted. The patient was then rotated carefully on his side maintaining the head in the same plane of the body, and a lumbar puncture was done. There was a complete block on the Queckenstedt test. The spinal fluid procured at this time was reported as xanthochromic but pale, and contained 250 mg. of protein. Shortly after lumbar puncture the sensory level for motion, position, and hypalgesia was at T4 dermatome, indicating a rise of approximately 5 segments.

A diagnosis of anterior spinal cord compression was made.

Operation. Cervical laminectomy was performed under intratracheal anesthesia with the removal of laminae C4, C5 and C6. The ligamentum flavum was completely



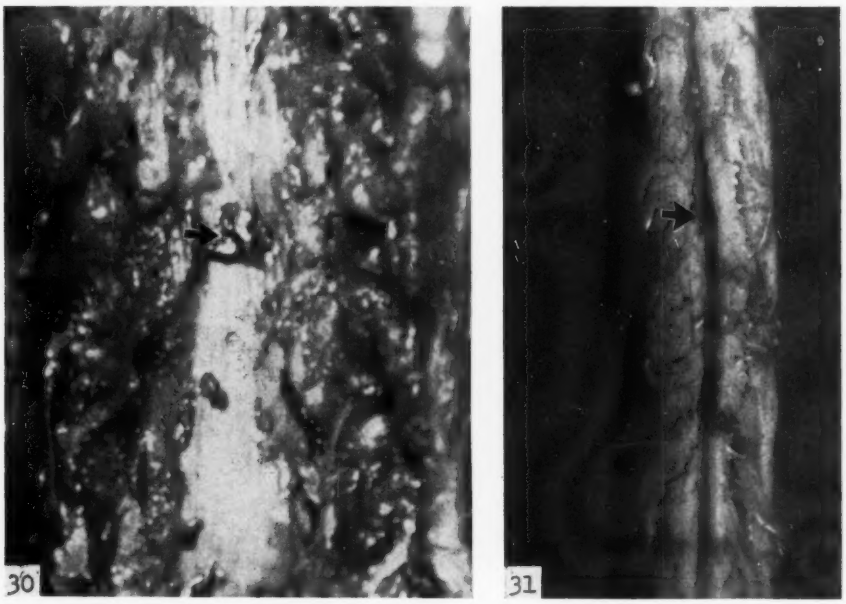
FIGS. 28–29. *Case 12.* (28) Fracture-dislocation at C5–C6. (29) Cervical spine overdrawn by skeletal traction without satisfactory realignment because of locking of facets. Reduction should have been accomplished at operation.

avulsed between C5 and C6, and C5 vertebral body was displaced anteriorly so that the tip of the spinous process of C5 came just to the level of the lamina of C6. After laminectomy the dura mater began to pulsate and bulge posteriorly. The dura mater was opened in the midline, and the arachnoid maintained intact. The arachnoid was incised laterally and the dentate ligaments were cut at three levels. At C5 the cord was markedly elevated and then dipped down anteriorly at the C6 vertebral level. The vessels and the cord itself appeared to be normal after sectioning the dentate ligaments and exploring anterior to the cord. There was no evidence of any herniated

disc. It was observed that after the dentate ligaments were cut the cord itself rose upward into the wound. The dura mater was left open. Cervical traction was maintained, but complete reduction of the fracture-dislocation was not accomplished because the facets were still locked (Fig. 29).

Course. A week after operation the patient's blood pressure dropped to 70/40, the pulse rate was 98, and his temperature was 102°. At 9:30 that morning his respirations became irregular, the blood pressure continued to fall and he was placed in a respirator. Transfusions were given to the patient. He complained of no pain but had a progressively downward course. His respirations ceased at 5 A.M. on March 27.

Necropsy. There was a subtotal extrusion of a disc into the spinal canal at C5-C6 interspace (Fig. 30). There were a few minute areas of contusion on the spinal cord at that level (Fig. 31). Acute purulent confluent lobular pneumonia was found and there were acute multiple abscesses in the prostate. Multiple acute gastric ulcers were discovered. The cause of death was listed as severe hemorrhage into the stomach from a bleeding ulcer.



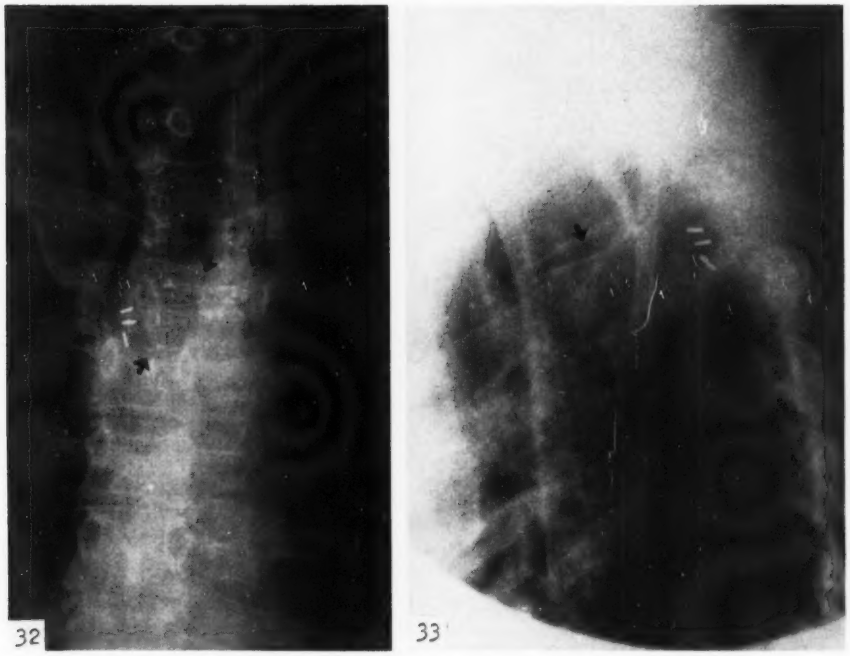
FIGS. 30–31. *Case 12.* (30) Postmortem appearance of anterior surface of spinal canal. Arrow indicates partially extruded cervical nucleus pulposus. (31) Cervical cord shown at necropsy. Arrows indicate the level of the cord that lay immediately above the extruded disc, demonstrating that there is little gross anatomical damage to the cord.

Microscopic sections of a portion of the cord that appeared normal grossly revealed extensive degenerative changes within the cord itself. Perivascular edema and extensive myelin degeneration of the cord were present.

Comment. This patient exhibited a progression of neurological signs from the time of accident until he entered the first hospital. On the basis of this,

operation was indicated. By the time he arrived at University Hospital after a trip of 60 miles without the benefit of traction, a true anterior cord compression syndrome had developed. The edema was very pronounced so that when lumbar puncture was performed, there was evidence of complete block, which was another indication for operation. If traction had been instituted during the trip to University Hospital, the block might not have been present. The case demonstrates how rapidly progression of neurological signs can occur in the face of complete block on lumbar puncture.

It should be noted that ulcerations of the stomach and duodenum have been reported in conjunction with injuries to the cervical area. The author is unfamiliar with any definite mechanism that might be the cause of such lesions.



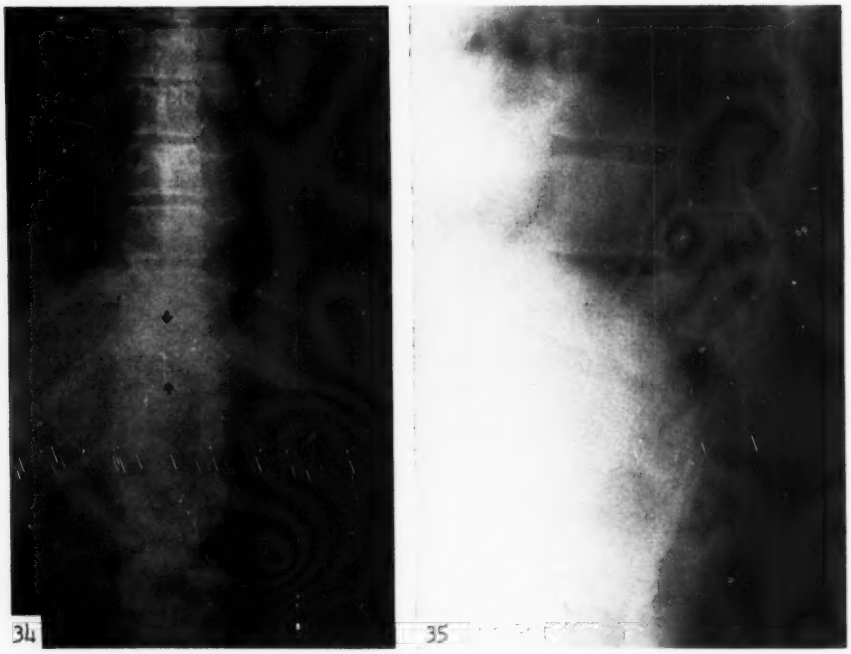
FIGS. 32–33. *Case 13.* Postoperative roentgenograms showing compression fracture and lateral displacement of T3 vertebral body on T4.

Case 13. H.H., a 33-year-old housewife, was involved in an automobile accident on the afternoon of July 13, 1953. She was thrown from the car and struck directly upon her back. She regained consciousness at a local hospital, but was suffering from traumatic shock.

Examination. By the morning of July 14, 1953 the neurosurgical consultant found a complete areflexic paraplegia except for an equivocal flexion of the right

great toe. There were hypalgesia and hypesthesia to the T4 dermatome but vibration, motion, and position sensations were retained over the entire body. Roentgenograms showed a fracture-dislocation of T3 on T4 vertebral body (Figs. 32 and 33), and T10 on T11 vertebral body (Figs. 34 and 35). Fractures of the right clavicle and scapula and the right transverse processes of L1, L2, and L3 were also demonstrated.

Operations. On July 17, 1953, the lamina were removed from T2 to T5, bilaterally. There was marked anterior compression and left lateral displacement of the spinal



FIGS. 34–35. *Case 13.* Postoperative roentgenograms demonstrating a compression fracture with minimal dislocation of T11 vertebral body.

cord by the T4 vertebral body. The T3 nerve root was sacrificed and a portion of the T4 vertebral body was chiseled away in order to accomplish decompression of the cord anteriorly and laterally.

On July 22, 1953, a complete laminectomy of T9 through T11 was performed. A portion of the fractured T11 vertebral body had to be removed anterior to the cord, and in order to accomplish this the right T10 nerve root had to be sacrificed. The dura mater was opened in the midline and the dentate ligaments were cut at two levels. The cord did not pulsate, but appeared to be normal. A catheter was passed upward with no evidence of blockage of the subarachnoid space. The dura mater was left open.

Course. By August 10, 1953 there was a drop in the level of hypalgesia from the T4 dermatome to T10, bilaterally. There was no movement of the lower extremities, but the Achilles reflexes could be obtained. On August 30, 1953, the patient could

move her left ankle, and within a week or two all movements had returned in the left lower extremity, but there was only minimal strength on the right side.

By July 1, 1954 the patient had improved markedly and was able to stand for a short time without the use of braces. Spasticity in her lower extremities had gradually diminished and her bladder control had almost entirely returned. However, about July 15, 1954, the left leg became numb around the region of the knee, and the left quadriceps muscle became weak. Strong burning sensations developed from the hips on downward and it was difficult to determine whether there was some increasing spasticity that was masking the strength in the left leg, or a true loss of motor power.

Comment. Originally the syndrome of anterior spinal cord injury was described merely in association with acute *cervical* spine trauma. It was thought that it would not be found in thoracic spine injuries because the force required to dislocate the thoracic spine necessarily must be very great to overcome not only the stability of the spine itself but also the added fixation effect of the thoracic cage. Because of the narrowness of the spinal canal, any such force causing disruption almost certainly would completely transect the spinal cord. In this case the patient's cord was apparently preserved because she struck very hard and flat on her back upon the ground. The facets were fractured at the two sites without acute hyperextension or acute flexion of the spine, thus permitting the laminae to be displaced dorsally, accomplishing decompression with preservation of the spinal cord. This patient unquestionably was aided by surgical intervention. If movement in the acute phase of convalescence had not irreparably injured the tightly compressed spinal cord, then chronic spinal cord compression still might have caused neurological deficit as more bone was laid down at the fracture sites (see Case 5).

CONCLUSIONS

1. The syndrome of anterior cervical cord injury is characterized by an immediate complete paralysis below the site of the lesion associated with a level of hypalgesia and hypesthesia consistent with the lesion, with preservation of motion, position and touch, and some vibration sense. In only 1 case was the syndrome not present immediately but evolved rapidly within a few hours (Case 12).

2. The anterior spinal cord injury syndrome may be associated with an isolated herniated cervical nucleus pulposus (Case 1), a herniated cervical nucleus pulposus in conjunction with a fracture-dislocation of the cervical spine (Cases 2 and 12) or fracture-dislocation alone (Cases 3, 4, 5, 8, 9, 10), and compression fracture of the cervical vertebra (Case 7). In 2 of the cases anterior spinal cord destruction was visualized at necropsy or at operation (Cases 6 and 11). The syndrome has been observed in only 1 case of thoracic injury and that was associated with a double fracture-dislocation (Case 13).

3. There was complete blockage on the Queckenstedt test in only 1 patient (Case 12). This patient was the only one who did not show the pure syndrome, for there was a marked progression of neurological signs immedi-

ately after injury and secondarily a block developed immediately after lumbar puncture. With the exception of this case none of these patients was operable under the usual criteria of the presence of a complete subarachnoid-space block or progression of neurological signs. *It is impossible to differentiate those patients who are suffering from anterior cord compression by a lesion, with the restriction of posterior cord displacement by the dentate ligaments, from those who have anterior cord destruction. Therefore, early operation is advisable, with section of the dentate ligaments and inspection of the interspaces for a surgical lesion.*

4. In general, myelography is contraindicated in this group of patients for fear of causing further damage to the already injured spinal cord.

5. These patients have been operated upon at varying intervals, ranging from 18 hours to 4 years after injury with varying degrees of recovery. The most striking result was observed in Case 7. This patient had not moved his extremities for a period of $5\frac{1}{2}$ weeks after injury, and yet 36 hours after cervical laminectomy with section of the dentate ligaments, he began to move his fingers and toes. This suggests that early operation is indicated to prevent prolonged anoxia caused by direct anterior spinal cord compression or possible compression of the anterior spinal artery.

6. When fracture-dislocation has occurred the "tear-drop fracture" (the name suggested by the anterior inferior surface of the vertebral body "dripping" anteriorly), "diver fracture," or "acute cervical flexion fracture" is common (Cases 3, 4, 5, 6 and 9). The posterior inferior margin of the same vertebral body in each of these cases had become displaced posteriorly into the spinal canal, causing anterior spinal cord compression or destruction. These patients should be placed immediately in skeletal tong traction upon an anterior Stryker or Foster frame. If the lesion is at the level of the phrenic segments C3-C4 (Case 10), or if there is respiratory difficulty, exploration should be deferred until this situation is stabilized. If the injury has occurred below C3–C4 level, decompressive laminectomy should be performed early with the patient in traction on the frame. A *carefully inserted* intratracheal tube should be used for anesthetization, and laminectomy should include three laminae. The dura mater should be opened in the midline and the arachnoid cut laterally so that the dentate ligaments may be sectioned at two or three levels. Transdural exploration may then be carefully made to exclude a herniated disc. If spinal cord swelling is marked, the dura mater should be left open, and the arachnoid, which has been left intact in the midline, will seal off the cerebrospinal fluid. After a period of 3 weeks in traction, cervical spinal fusion should be performed by wiring together the proximal and distal spinous processes, inserting a carefully shaped, well fitting roof bone graft bilaterally to cover the surface of the cord. Bone chips may then be placed laterally over the roughened facets. After 8 weeks the tongs should be replaced by a Forrester collar with a back brace.

Decompression with removal of three laminae is necessary to permit

sufficient space for section of dentate ligaments at two or three levels and exploration anterior to the cord. A limitation of the laminectomy to two laminae in order to pacify orthopedic colleagues so they need perform a less extensive fusion could readily have cost the life of the patient in Case 6. The posterior surface of the cervical spinal cord appeared grossly normal in this case at operation, but a postmortem examination 5 days later revealed a constriction at the level where the C4 lamina had compressed the posterior surface of the spinal cord. Swelling of the cord from traumatic handling may have resulted in compression, hypoxia and further central cephalad destruction, thus destroying the phrenic segments at the C4 level and causing the patient's death.

7. Spinal fusion is a necessity in the cases in which there is "tear-drop" or "acute flexion fracture-dislocation" with a partially damaged cord, for even after keeping patients in traction 12 weeks (Case 7) there may be slippage with the possibility of destroying or severely injuring the cord (Cases 3, 4, and 6). Case 4 demonstrated that although the cervical roentgenograms suggest a solid fusion, only a fibrous union with pronounced spinal instability was found at the time of operation 4 years after injury.

8. Some patients may recover some degree of function spontaneously from anterior cervical spinal cord compression (Case 5). However, there is always danger of chronic arthritic spurring at the level of the fracture-dislocation, causing chronic anterior cord compression and resulting in spasticity (Case 5) requiring operation at a later date. The author firmly believes that it is advisable to operate upon these patients early in order to decompress the cord and permit it to ride freely over any bony defect. Also if there is an associated herniated disc present at the time of the acute injury, it is more readily removed and does not become calcified to cause chronic anterior cord compression. Stabilization of the spine by spinal fusion prevents movement and continued irritation to the bone, preventing calcium from being laid down anterior to the cord.

SUMMARY

In summary, in acute trauma to the spine there is a syndrome of acute anterior spinal cord injury which is characterized by an immediate complete paralysis with hypesthesia and hypalgesia to the level of the lesion, with preservation of touch, motion, position and part of vibration sense. This syndrome may be caused by acute anterior spinal cord compression or destruction of the anterior portion of the cord. Since a surgical lesion causing spinal cord compression is indistinguishable from a nonsurgical destructive one, these patients should have an exploratory laminectomy and the majority of them should have a spinal fusion secondarily 2 or 3 weeks later. Emphasis should be placed upon the fact that these patients have attained their maximum neurological deficit and they do not present the usual criteria for surgery, namely, progression of neurological signs or evidence of blockage

of the cerebrospinal fluid on the Queckenstedt test. Thirteen cases are presented in detail to illustrate the pathological conditions producing this neurological pattern.

The author wishes to express his gratitude to members of the Bone and Joint Department, University Hospital, Ann Arbor, under the direction of Dr. Carl Badgley, who have generously aided in consultation and performed all of the spinal fusion operations on patients treated there.

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COMPRESSION RATHER THAN DECOMPRESSION FOR TRIGEMINAL NEURALGIA*

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CONSIDERABLE progress has been made during the past 4 years in the surgical management of major trigeminal neuralgia. However, very little has been added to our meager knowledge concerning the etiology of the pain.

Woltman in 1951 suggested to us the possibility of controlling facial neuralgia by decompression of the peripheral branches of the trigeminal nerve at their points of exit from the skull. This suggestion was based upon the experience of Woltman and Williams[2] in treating facial tic by decompression of the seventh nerve.

During the period from 1951 to 1953, 10 patients were surgically treated for trigeminal neuralgia by decompression of the peripheral branches of the trigeminal nerve at the foramen ovale and foramen rotundum.

The surgical procedure consisted of temporal craniotomy, exposure of the foramen ovale and/or foramen rotundum, enlargement of the foramen by means of a motor-driven high-speed dental drill, neurolysis of peripheral branch or branches with saline followed by multiple linear incisions in the sheath of the nerve (Fig. 1).

Every patient experienced complete relief of pain without significant objective impairment of facial sensation. Several patients noted mild subjective differences in tactile sensation, but this difference seldom could be demonstrated objectively for longer than a few days postoperatively.

The results were gratifying but the mechanism of relief was difficult to explain. During this period it was learned that Taarnhøj in Copenhagen was accomplishing equally satisfactory results by decompression of the posterior root of the trigeminal nerve. His subsequent publication[1] verified the reports that we had obtained, but it was difficult to understand how the two procedures, both decompressing the fibers of the nerve, but at widely separated points, could accomplish the same results.

Numerous observers concluded after Taarnhøj's report that the causative factor of trigeminal neuralgia was compression of the posterior root. If such were the case, how could decompression of one or more branches peripheral to the ganglia afford complete relief of pain and no loss of facial sensation?

The experience gained from our first 10 patients was presented in 1952‡

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‡ American Academy of Neurological Surgery, New York City, October 1, 1952.

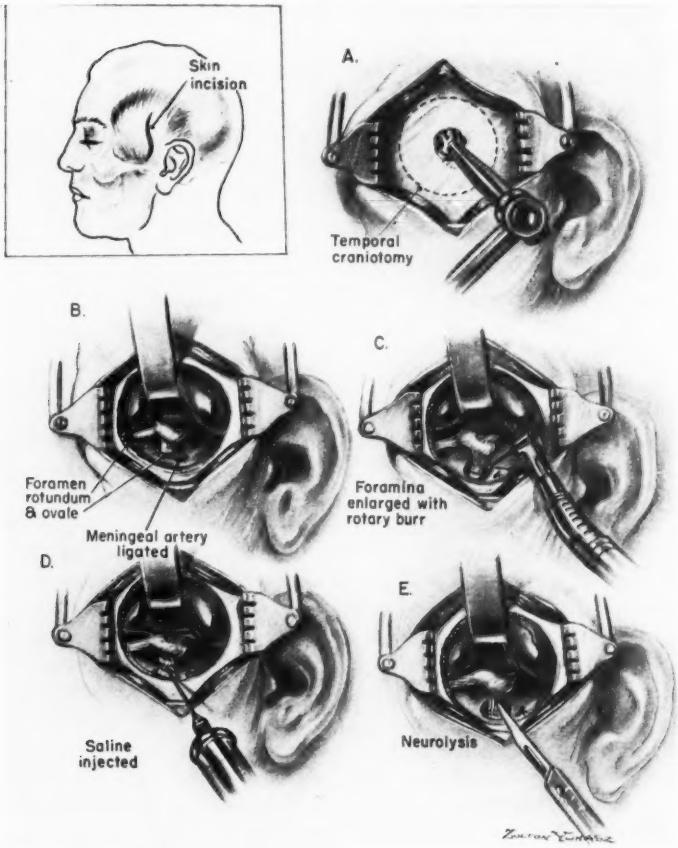

FIG. 1. Technic employed in foraminal decompression.

at which time we considered the results as excellent. In spite of the gratifying postoperative course of these patients, publication of our results was withheld because the period of postoperative observation was considered inadequate. Periods of complete relief of pain following alcohol injection may extend over 3 or 4 years.

In reviewing our results 3 years after foraminal decompression we were impressed with the high percentage of lasting results. The mechanism of relief of pain remained obscure, but if relief could be obtained from decompression of either the peripheral branches or posterior root the two methods must have something in common. It also seems obvious that the etiological factor producing trigeminal neuralgia could not be limited to the posterior root. This assumption is supported by the fact that no one has ever found any

constrictive type of organic pathology in the posterior root, nor ever found a dural canal at operation too small to accommodate the posterior root fibers adequately. Furthermore, electrical stimulation of the posterior root does not produce typical paroxysms of pain.[3]

The only apparent feature common to both procedures was that of operative trauma. With this in mind, our first patient was operated upon in December 1953 using the technic of posterior root compression (Fig. 2). The procedure consisted of the usual temporal approach with the patient in the sitting position. The dura mater was reflected from the dura propria in the usual manner, using a small dental roll. The posterior portion of the ganglion and adjacent part of the posterior root were exposed. A small vertical opening was made in the dura propria near the ganglion, thus exposing the posterior root fibers. Care was taken not to expose more than a minimal portion of the posterior root, so that the procedure could in no way be considered as the usual decompression technic.

After the fibers were exposed they were gently compressed with a small portion of a dental roll if the patient's pain involved the second and third divisions. If the patient's pain was limited to either division alone, a small blunt instrument was used to compress selectively the appropriate fibers of the root. More recently the back of a blunt dissector has been used as the instrument for compression.

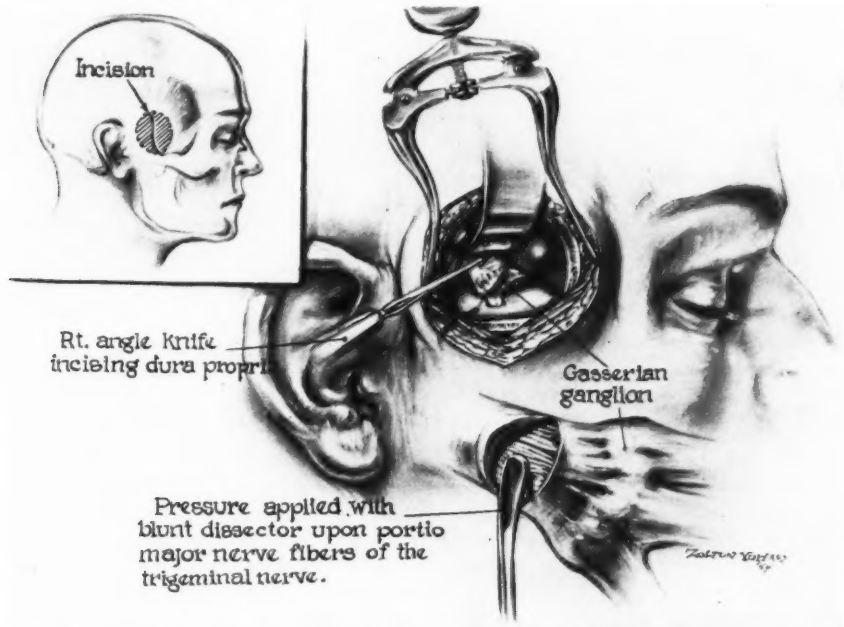

FIG. 2. Operative exposure and method of compression of the posterior root fibers.

The compression technic has been employed in operating upon 29 patients. All have been completely relieved of their preoperative pain. None has had postoperative facial anesthesia. The majority of patients were aware of some subjective difference in sensation on the two sides if the face was rubbed, but this difference was minimal and of short duration. Five patients have had objective impairment of sensation, particularly of the lower lip, which was charted as being −1 to −2. The sensory changes gradually disappeared except in 2 patients. These 2 patients stated that the facial sensation has returned almost to normal but actually the minimal objective changes persisted. The corneal reflex was normal in all patients in this series. Function of the muscles supplied by the motor branch was retained in every patient.

This is a preliminary report of a simplified method of surgically treating trigeminal neuralgia. It would appear that compression of the trigeminal fibers during operation is the factor that has afforded relief of pain with the socalled decompression procedures.

Careful observation of these patients over a period of several years will be necessary to determine the true value of this procedure. However, the compression operation affords the simplest method of relieving the pain of trigeminal neuralgia and preserving facial sensation without the morbidity that accompanies other methods.

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AQUEDUCT STENOSIS

CLINICAL ASPECTS, AND RESULTS OF TREATMENT BY VENTRICULO-CISTERNOSTOMY (TORKILDSEN'S OPERATION)

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PARTIAL or complete occlusion of the sylvian aqueduct may be caused by congenital malformation,[3,16,27,28] post-inflammatory ependymitis,[1,28] or may result from pressure of an intracranial tumour,[22,23,24] aneurysm[5] or angioma.[10] In order to overcome such an obstruction some new pathway must be made that will enable the cerebrospinal fluid to gain access to the subarachnoid space covering the cerebral hemisphere or be directed to some other absorptive space.

The obvious method would be to dilate the aqueduct or to insert a tube connecting the third and fourth ventricles. Dandy,[3] in 1920, reported 2 cases in which he passed a tube from the fourth ventricle through the aqueduct. In both cases the tube was later removed. One patient died and the other was reported as being well 1 year after operation. Fraser and Dott[9] reported 5 cases of aqueduct stenosis, in 4 of which the operative procedure was a cerebellar decompression and insertion of a probe through the aqueduct. One patient was stated to be cured, 1 improved, and the other 2 died. The follow-up period was not given. Leksell,[18] more recently, used a tantalum wire coil to pass through the stenosed aqueduct. He had 2 operative deaths, and 1 death a year after operation, in a group of 10 patients. Three infants under the age of 1 year did not survive the operation.

Shelden, Parker and Kernohan[31] described the treatment of 6 patients with aqueduct stenosis, 1 with an associated meningocele. All 6 died: 1 after lumbar puncture, 3 after cerebellar decompression, 1 after subtemporal decompression and 1 after third ventriculostomy.

Stookey and Scarff,[32] summarising the results of treatment in reported cases of the condition, found that there were 10 deaths among patients not submitted to operation, 1 death after subtemporal decompression and puncture of the corpus callosum, 2 deaths following ventriculography, 2 deaths after suboccipital decompression, and 1 death 5 weeks after this procedure. No survivals were recorded. These authors then reported the results of their operation of third ventriculostomy. Four patients were submitted to this procedure. One died after 1 month, but the other 3 were stated to be well 6 months, 14 months and 3 years later. Here at last seems to be a method of treatment that yields successful results.

Pennybacker[25] in 1940 reported to the Royal Society of Medicine 18 cases of aqueduct stenosis. A variety of operations had been practised on

these patients. Cerebellar decompression had been performed in 7, 1 of whom died postoperatively from a wax embolus. Another patient had a recurrence of symptoms 3 months later. Three patients were well 3 years after operation and one was well 4 years after operation. Two of these patients had been subjected to a course of irradiation. The final patient, who had had a pineal exploration in addition to the decompression, was well 4 years later. Third ventriculostomy was performed in 5 patients, 2 of whom died postoperatively whilst the other 3 were reported as being well 4 months, 1 year and 2 years later. In 2 patients the corpus callosum was split but 1 died postoperatively and the other 10 months later. The only patient subjected to a pineal exploration and irradiation was well 7 years later. One patient who had had a subtemporal decompression and a course of X-ray therapy was still symptom-free after 6 years. One patient died from an intracranial infection after ventriculography and the remaining patient died 3 years after diagnosis, having received no operation.

Drainage of the cerebrospinal fluid into the body tissues or cavities has been advocated by many, tubes of varying construction having been passed between the ventricular system above the stricture to the mastoid,[21,33] jugular vein, the soft tissues of the neck or scalp, peritoneum and pleura, to mention but a few.[6,7,11,14] No one of these methods has proved satisfactory and the death rate has been high. The tendency of cerebrospinal fluid to produce a dural membrane around itself prevents success by many of these methods whilst drainage into such cavities as the middle ear opens a potential road for infection of the subarachnoid spaces.

Avulsion of the choroid plexus to provide a pathway from the lateral ventricle into the cisterns around the brain stem was advocated by Hildebrand in 1904.[15] Hyndman[13] described 1 case of stenosis of the aqueduct treated by his operation of disruption of the choroid plexus but the patient died 2 months later.

Third ventriculostomy, as mentioned above, was one of the early methods of treatment that gave some hope of success in the management of patients with aqueduct stenosis. Stookey and Scarff[32] first reported 4 operations with 3 survivals. Their original procedure required the making of an opening in the lamina terminalis above the optic chiasm, the exposure being through a transfrontal craniotomy. Scarff[30] later reported on 10 cases, in which 7 operations were performed for the relief of aqueduct stenosis. There were no operative deaths but 1 patient died a month after surgery. Another survivor died 5 years later. Three other patients were living 1, $3\frac{1}{2}$ and 5 years after operation. The follow up was incomplete for the remaining patient. White[36] gave his results of this operation on 3 patients with occlusion of the aqueduct. Two survived but 1 died 2 months after operation from pneumonia. Krayenbühl, Werner and Martin[17] performed this operation on 17 patients, 4 of whom had stenosis of the aqueduct. Of this latter group 3 were relieved of symptoms, for 3 years in 2 cases and 4 years in 1. The fourth patient was well for 3 years but then suffered a recurrence of symptoms.

Scarff was not entirely satisfied with the results of the operation of anterior third ventriculostomy and in 1950[29,30] described a modification in which the third ventricle was opened anteriorly and then a further opening was made into the floor of the ventricle, so allowing the cerebrospinal fluid to escape into the interpeduncular cistern. This was an attempt to prevent the formation of the frontal subdural collection of cerebrospinal fluid that accumulated after the original procedure. He reported 17 cases of aqueduct stenosis treated by this new method in a series of 34 patients. In this series 4 patients died postoperatively and 1 died 6 months after operation. Of the remaining 12, 11 were relieved of their intracranial hypertension for periods varying from 2 months to 14 years. Only 1 survivor was not relieved by operation.

Dandy[4] had earlier experimented with the operation of third ventriculostomy, his method being to puncture the posterolateral wall of the third ventricle through a subtemporal approach. He reasoned that the falling back of the medial portion of the temporal lobe would close the cisterns around the brain stem, and so prevent the collection of subdural cerebrospinal fluid. He reported 1 operative death and 3 later deaths at 3, 5 and 6 months. Seven patients required reoperation following closure of the opening into the third ventricle. Twenty-nine patients survived more than 1 year, the longest follow up being $23\frac{1}{2}$ years. He also reported 63 patients under the age of 1 year with 10 hospital deaths and continued survival in 21 cases.

Ventriculo-ureterostomy has been used by Matson[20] to treat 2 children with aqueduct stenosis. Both were improving $2\frac{1}{2}$ and 4 months after operation.

It was in 1937 that Torkildsen first performed his operation for providing a new sylvian aqueduct by passing a soft rubber tube from one lateral ventricle, through a subgaleal tunnel over the occiput, and inserting the lower end into the cisterna magna. Ten years later[34] he published his results in a series of 47 cases, in 13 of which the patients were suffering from aqueduct stenosis. Four of the 13 died postoperatively, whilst 2 others, at 6 weeks and 4 months respectively, succumbed from the continuation of the raised intracranial pressure in spite of the short-circuit operation. One more patient died $2\frac{1}{2}$ years after operation, following ventriculography, although she had remained in good health from the time of the operation. Thus over half of Torkildsen's original 13 patients failed to survive.

Fincher, Strewler and Swanson[8] reported 5 patients treated by Torkildsen's operation of whom but 1 had died postoperatively, whilst the rest were relieved and alive 18 to 36 months later.

Of the 4 patients described by Herlin[12] 2 died postoperatively, 1 was alive but unrelieved 5 years afterwards, whilst the remaining patient was surviving and well 10 months after operation.

This paper presents a series of 25 patients treated by Torkildsen's method for non-neoplastic aqueduct stenosis and outlines the clinical features, radiological findings and results of treatment.

The series is too small to subdivide into differing pathological groups, were it possible to do this with certainty in the absence of pathological proof. The causes, therefore, probably include many varieties of stenosis of the aqueduct of Sylvius, with the exception of tumour. These have been described as absence, obliteration, partial atresia, duplication, forking, and occlusion caused by ependymitis. The only case of a septum of the lower end of the aqueduct, seen in this clinic, has not been included, as the operative procedure consisted of rupture of this membrane through the fourth ventricle.

CLINICAL PICTURE

Age at Onset of Symptoms. In about two-thirds of the patients symptoms developed before the age of 20 years: 9 before the age of 10, and 8 between the ages of 10 and 19 years. Of the remaining 8 patients, symptoms commenced in the third decade in 5, in the fourth decade in 2 and in the fifth decade in 1 (Table 1).

TABLE 1

Age at onset of symptoms

0 to 10 years	9
10 to 20 years	8
20 to 30 years	5
30 to 40 years	2
42 years	1

Duration of Symptoms before Treatment. There was a very wide variation in the duration of symptoms, so far as this could be estimated, the shortest history being only 3 weeks and the longest 46 years. Eleven patients (44 per cent) gave a history of less than 1 year and in 8 of these cases the duration of symptoms was less than 6 months. The remaining 14 cases were divided into small groups giving histories of longer periods of disability and are set out in Table 2.

The age at onset of symptoms might be a point in differentiating congenital from acquired lesions, the latter starting later in life, but we are not

TABLE 2

Duration of symptoms

0 to 6 months	8	Under 1 year: 11
6 to 12 months	3	
1 to 2 years		2
2 to 3 years		2
3 to 4 years		3
4 to 5 years		2
5 to 10 years		2
10 to 20 years		1
20 to 30 years		1
46 years		1

able to make any such deductions from this series. The duration of symptoms could be an indication of the degree of obstruction of the aqueduct—the less the stenosis the longer the period the patient is able to tolerate the cerebrospinal fluid obstruction. By the time these patients come to ventriculography, the stenosis appears to be almost complete in all, and so, again, we are unable to comment on this feature.

Age of Patient at Operation. This ranged from 18 weeks to 58 years. Four patients were under 10 years, 10 aged between 10 and 20 years, 5 between 20 and 30 years, 3 from 30 to 40 years, 2 in the fifth decade and one aged 58 years.

There is no doubt that the very young patients with aqueduct stenosis would not be included in this series, the operation of ventriculocisternostomy not being practised in young children. Matson[20] regards extreme youth as a contraindication to the performance of Torkildsen's operation, stating that the subarachnoid spaces are imperfectly developed and will not allow the passage of cerebrospinal fluid through the normal channels over the vertex. Dorothy Russell's[28] researches into hydrocephalus have shown that many infants died from hydrocephalus caused by aqueduct stenosis. Furthermore, stenosis of the aqueduct may be associated with other congenital abnormalities and so make the problem of treatment in early life a complex one.[16,19,26,27] Certainly the patient, in this series, who was operated upon before the age of 1 year, died in the immediate postoperative period.

SYMPTOMS

Headaches occurred in 22 patients, but in only 12 could it be said to have been the presenting symptom. In this latter group the headaches had been present for less than 7 months in 8 patients. In one instance the patient had had three severe headaches in 4 years but a persisting headache for 5 weeks before admission. Another had had a mild headache for 2 years but more severe headache in the 5 months before admission. In the other 2 the headaches had been the main symptom for 1 and 6 years respectively.

In the group in which the headache was an incidental symptom it had been present for less than 6 months in 3 cases, 9 months in 1 and 16 months in another. One patient recalled an episode for 1 week, 2 years earlier. Two patients described occasional but not severe headaches for some time and the final patient could only describe a feeling of fullness in the head associated with nausea and vomiting.

The headache usually had the features of that of raised intracranial pressure, worse in the morning on waking, aggravated by exertion, often associated with nausea and vomiting, and usually clearing during the day. The site was variously described as "all over," bifrontal, bitemporal or vertical but never occipital. The intermittent nature of the headache was apparent in those patients with the longer histories, and the intervals between the attacks might vary from a few weeks to many months. As noted above, there were instances of many years of freedom from headaches. In such

cases the paroxysms of headache suggested temporarily increased obstruction of the cerebrospinal fluid pathways with subsequent re-establishment of the flow. Those patients with headache of but a few weeks' or months' duration before admission to hospital all complained of the persistent ingravescent nature of the pain suggesting that a final obstruction or failure of compensation had taken place.

Nausea and Vomiting. These, the next most common complaints, were present in 13 of the 25 patients, and in 12 of the 13 were associated with the bouts of headache although they did not necessarily occur with every attack of pain in the head. In the remaining case there was no complaint of headache at all, vomiting being an isolated symptom.

Visual failure was a spontaneous complaint in 11 cases and in only 1 other patient was there visual deterioration on examination. Visual failure had been noticed for 2 weeks in 2 patients, 2 months in 2, 4 months in 1 and 18 months in 2 others. One patient had had deterioration for 2 years, another for 3 years and another for 4 years. The final patient complained of failing vision for 20 years. Two patients had early papilloedema, 3 moderate swelling, 3 severe swelling and 3 had secondary optic atrophy. Except that the patient with a 20-year history had secondary atrophy, there appeared to be no relation between the length of history and the degree of papilloedema.

Mental Defect or Deterioration. This condition was present in 11 cases but was never the primary complaint of patients or relatives. Mental deficiency was of minor degree in 5 patients and moderate or severe in the other 6. The mental change in 2 of these 6 patients was not in evidence until after a head injury which was severe enough in 1 instance to produce a hemiplegia.

Unsteadiness occurred in 10 patients and clinical examination in 9 of these revealed incoordination or ataxia of the limbs suggestive of cerebellar dysfunction. In the tenth case nystagmus was present with minimal pyramidal signs. In 4 instances the unsteadiness was the presenting symptom and had been present for 2 weeks in 1, 2 years in 1, 6 years in 1 and for an unstated number of years in the fourth.

Increasing weight was complained of by 6 patients and in 2 of these there were associated endocrine changes.

Endocrine changes occurred in 5 patients, 2 females having irregular periods, 2 males being sexually underdeveloped, and menstruation not having occurred in the fifth, a girl of 15. Endocrine changes may well be associated with hypothalamic disturbances produced by the dilated third ventricle as are known to occur in patients with tumours of the posterior part of the third ventricle or with obstruction of the ventricular system more distal to this.

Attacks of unconsciousness occurred in 5 patients. In 1 case such attacks were the presenting symptom and were sometimes associated with generalised convulsive movements: this patient had suffered from headache and vomiting for a number of years. In another instance the attacks appeared to be epileptiform following a severe head injury at the age of 18 months. In a

third case the attacks also appeared to be those of post-traumatic epilepsy. The other 2 patients had intermittent attacks of unconsciousness, 1 having had two attacks in 2 to 3 weeks only, and the other a number of "faints." In only one instance, therefore, were these attacks suggestive of an acute hydrocephalic episode.

Diplopia was a complaint of 5 patients, all with severe headache of brief duration, and in 3 instances was one of the presenting symptoms. Three patients had a unilateral sixth nerve palsy but no cause could be found in the other 2.

Epilepsy was an associated symptom in 4 patients, 2 of whom have been mentioned above. The other 2 had attacks of dissociation suggestive of temporal lobe seizures.

Giddiness was complained of by 4 patients and in 1 was associated with unsteadiness and cerebellar signs. None of the other 3 had clinical evidence of cerebellar or brain stem dysfunction. In 1 instance the vertigo was a presenting symptom.

A large head was remarked upon in 3 cases, and in 1 child aged 18 weeks was the only symptom.

A weak arm or leg as part of a hemiparesis was present in 3 patients but in all could be attributed to natal or postnatal trauma.

Incontinence was present in 3 patients.

Paraesthesiae of the arms or legs were present in 3 cases, in only 1 of which could the sensation be attributed to epilepsy.

Dysarthria was present in 2 patients, both with well marked cerebellar signs.

"*Bad nerves*" had been complained of by 2 patients for a varying length of time.

Difficulty in opening her mouth for the previous 4 months was the surprising symptom presented by 1 patient, a nurse.

PHYSICAL SIGNS

Fundus Examination. Bilateral papilloedema was present in 16 patients, unilateral papilloedema in 1 and secondary optic atrophy in 4. Thus in 21 cases there was evidence that raised intracranial pressure was present or had been present. The papilloedema was described as early in 5 cases, and thus in 16 patients incontrovertible fundus changes were present. Two patients were said to have primary optic atrophy and the remaining 2 to have normal fundi. These 2 were aged 18 weeks and 10 years with histories of 5 weeks and 14 months respectively.

Visual Acuity. Seven patients had an acuity of 6/60 or less in one eye, 1 patient had a bilateral cataract and 1 was too young for visual assessment.

Cerebellar Signs. Ataxia of the arms or legs occurred in 14 patients. Nine of these had complained of the unsteadiness as a symptom. In the other 5 the cerebellar signs were of slight degree only.

Nystagmus was present in 7 patients and was unassociated with other

cerebellar signs in only 2. One of these made a spontaneous complaint of giddiness.

Pyramidal signs, either as a manifest hemiplegia or as a reflex change only, was present in 11 cases. In 1 case an extensor plantar response was the only abnormality and in 2 other cases, an increase in the tendon reflexes on the abnormal side. In 1 instance the hemiplegia dated from birth, in another from a head injury at 18 months, and in 1 it followed an illness at the age of 18 months (? encephalitis). The hemiplegia in the remainder of the patients was only slight.

Facial weakness as the only sign of an upper motor neurone lesion was present in 3 other patients.

Obesity was an observed feature in 12 patients and included all those with endocrine disturbances.

A large head was recorded in 8 cases, although it had been commented upon by the relatives in 3 only. In 7 of these patients the onset of symptoms was before the age of 12 years. The other patient was 30 years old on admission and her history was of 2 years' duration.

A sixth nerve palsy was unilateral in 5 cases and bilateral in 1. Five of these patients had severe papilloedema and 1 had secondary optic atrophy.

Deafness was present in 3 cases.

Visual field defects were present in 2 patients, nasal in one eye in 1 patient and bitemporal in the other. Constriction of the visual fields and enlargement of the blind spot were common findings.

Café au lait patches were present in 3 patients, 1 of whom had generalised neurofibromatosis. Pennybacker[25] reported a similar patient.

Hyposmia, diminished corneal reflexes and ptosis were each recorded once only in the clinical examination of the patients of this series.

Loss of position sense, other than in association with a severe hemiplegia, occurred in the arm and leg of the same side of 1 patient.

An *occipital naevus* was present in 1 case.

Comment. The clinical picture of aqueduct stenosis is not, therefore, clearly defined and, indeed, many writers in the past have brought attention to the confusing array of symptoms and signs suggesting a diagnosis of cerebellar tumour.[2,22] To us there appears to be a group of patients in whom a preventriculographic diagnosis of aqueduct stenosis can be entertained. It is composed of children of 10 to 15 years of age, usually a little slow at school and overweight, who complain of increasing headaches over a period of a year to 18 months. They may or may not have noticed visual deterioration; the parents may comment on the size of hat the child takes. Examination discloses a head on the large side of normal with a high-pitched percussion note. Bilateral papilloedema of slight to moderate degree is present, a convergent without demonstrable weakness of eye muscles is common. There is a fine tremor of the outstretched hands with a little ataxia of movements. Balancing tests are not well performed. In these patients the history is too long and the signs are too slight to make a diagnosis of cerebellar tumour,

the slightly large head and the abnormal percussion note indicate that the hydrocephalus has been advancing for a longer period than the history suggests, and one is faced with a patient of preadolescent age with a benign obstructive hydrocephalus without any certain localising signs but with questionable cerebellar dysfunction. Smallness of the posterior fossa on palpation will weigh heavily in favour of the diagnosis of aqueduct stenosis. Of course diagnosis can be made by pneumography with that certainty necessary for operation.

RADIOLOGICAL EXAMINATION

Plain roentgenograms of the skull demonstrated raised intracranial pressure in 20 instances. Erosion of the dorsum sellae and digital markings in the vault were the common findings (Fig. 1). In 4 instances the posterior fossa

FIG. 1. Roentgenogram showing suture diastasis, erosion of the dorsum sellae, and increased convolutional markings in the skull of a patient with aqueduct stenosis.

was small and suggested the diagnosis of aqueduct stenosis (Figs. 2 and 3). Dandy[4] stated that the inion is situated at a relatively lower level in those patients who have hydrocephalus caused by aqueduct stenosis, than when the hydrocephalus is produced by any other cause. It is probably more nearly correct to say that a benign obstruction of the ventricular system between the third ventricle and the fourth ventricle, commencing in childhood, is associated with a greater relative enlargement of the supratentorial por-

FIG. 2. Roentgenogram of the skull of an adult patient with aqueduct stenosis, showing the small posterior fossa.

FIG. 3. Roentgenogram of the skull of a child with aqueduct stenosis, showing the enormous hydrocephalic enlargement of the vault but without enlargement of the posterior fossa.

tion of the skull than if the obstruction lies in or below the fourth ventricle. A posterior fossa tumour of benign nature will be associated with an expansion of the posterior fossa caused by the presence of the tumour, as well as enlargement of the supratentorial portion of the skull vault caused by the hydrocephalus. The inion thus remains in a relatively high position. One skull in this series, that of a young child, showed a very great increase in the size of the head, separation of the sutures and enlargement of the fontanelles. One other patient had an associated platybasia.

Another feature of a nonspecific nature, frequently seen in the plain roentgenograms, is the presence of large diploic channels in the occipital bone

FIG. 4. Roentgenogram of the skull of a patient with aqueduct stenosis showing the large diploic channels in the occipital bone over the posterior fossa.

over the posterior fossa. Longstanding raised intracranial pressure is the probable cause of the opening up of these venous pathways. Fig. 4 shows a skull with these diploic channels, in this instance over the supratentorial portion of the skull as well.

Ventriculography was performed in 24 cases, the contrast media being air only in 12, myodil only in 5, and both air and myodil in 7. It is our practice at the present time to use myodil alone or myodil plus 10 cc. of air into the ventricles of a patient suspected of suffering from aqueduct stenosis. If at ventriculography the lateral ventricles are large, then 1 cc. of myodil (pantopaque, ethiodan) and 10 cc. of air are introduced into the ventricles. If the ventricles are small, then our suspicions were probably ill founded and we proceed to ventriculography with air in the usual manner. Another method which we have found useful is to introduce only 10 to 15 cc. of air into

the ventricles if they are large. This air can then be manipulated into the third ventricle and aqueduct and will not be obscured by overlying air in the temporal horns, as would be the case if larger amounts of air were used. Verbiest[35] obviously found the same difficulty for he suggested manipulating air into the third ventricle and then replacing the air in the lateral ventricles with saline before taking the X-rays!

Ventriculographic Appearances. The first and obvious feature is the enlargement of the lateral and third ventricles. This at once suggests that the obstruction of the ventricular system is at or below the posterior portion of the third ventricle. We have found a number of different appearances of the stenosed portion of the aqueduct:

(1) Partial Occlusion. Fig. 5 shows a narrowing of the aqueduct just after its origin from the third ventricle, occurring in a child 5 years old.

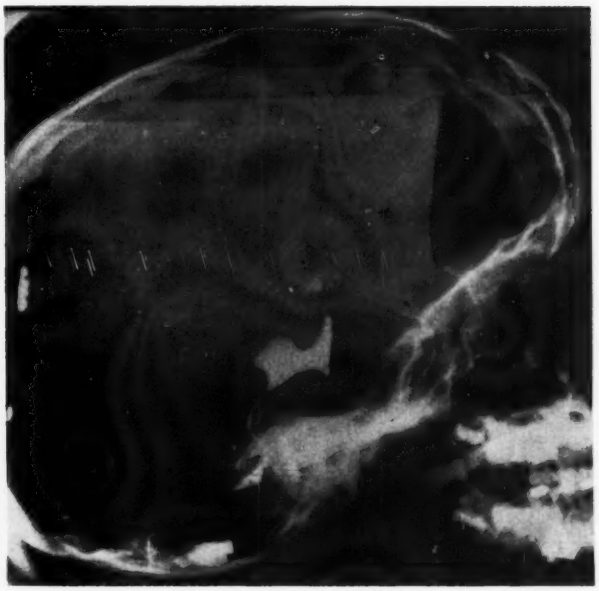

FIG. 5. Myodil ventriculogram showing partial occlusion of the sylvian aqueduct.

(2) Funnelled Aqueduct. Fig. 6 demonstrates a similar appearance of the proximal portion of the aqueduct, but in this case myodil could not be made to pass through the distal portion. The pointed ending is well shown.

(3) Bulbous Type. Fig. 7 shows an obstructed aqueduct ending in a bulbous fashion as though enlarged by the occlusion at the lower end. Fig. 8 is an air ventriculogram showing a similar appearance.

(4) Atresia of the Aqueduct. Fig. 9 shows the appearances of the myodil ventriculogram when none of the opaque material can be passed through the

FIG. 6. Myodil ventriculogram to show funnelling of the aqueduct of Sylvius.

aqueduct. The projection of the third ventricle toward the mouth of the aqueduct is to be seen in the upper blob of myodil.

No anteroposterior projections are shown, but are necessary to confirm that the posterior portion of the third ventricle and the aqueduct and fourth ventricle are not displaced to one side. Projections into the third ventricle as from pineal tumours or thalamic tumours should be easy to distinguish. Tumours of the posterior fossa will show kinking with or without lateral displacement of the aqueduct and may show displacement of the fourth ventricle. Brain stem tumours do not cause obstruction of the aqueduct without deformity of that structure. The first three types of ventriculo-

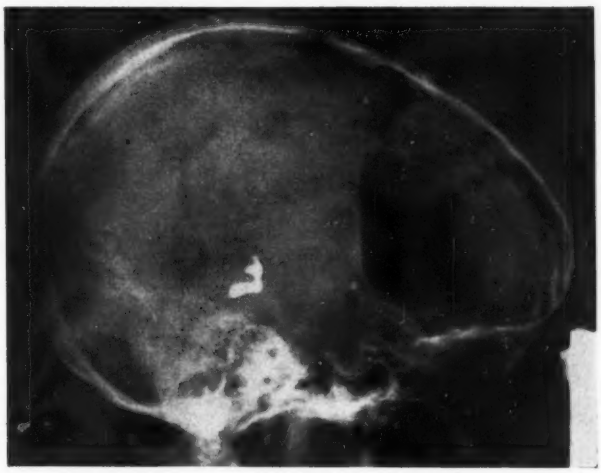

FIG. 7. Myodil ventriculogram showing the bulbous proximal portion of the aqueduct.

FIG. 8. Air ventriculogram to show enormously dilated lateral ventricles and the bulbous ending of the occluded aqueduct.

FIG. 9. Myodil ventriculogram showing the opaque medium in the posterior portion of the third ventricle but with none passing through the aqueduct.

graphic appearances mentioned above are pathognomonic of stenosis of the aqueduct provided that there is no anteroposterior or lateral displacement of that structure seen on the ventriculograms. The fourth type may give doubt in diagnosis as without visualisation of the aqueduct or fourth ventricle it is impossible to exclude a tumour as the cause of the occlusion.

In this series, the only patient not submitted to ventriculography was one in whom a posterior fossa tumour was suspected clinically. After a negative cerebellar exploration, a Torkildsen's operation was performed.

OPERATIVE PROCEDURE

Ventricular Drainage. In 4 cases, catheter drainage of one lateral ventricle was established, 2, 4, 5 and 6 days respectively, before operation. These patients were all deemed to be bad operative risks because the severity of the headache was suggestive of an acute hydrocephalic attack. Albeit 2 of these patients died after the ventriculocisternostomy and we are not able to say whether this was in spite of, or because of, the preliminary decompression.

Anaesthesia. General anaesthesia was used in every case in the series and differed in no way from that in use in the departments concerned with most major neurosurgical procedures. The induction has been that appropriate to the age and condition of the patient followed by nitrous oxide and oxygen with such adjuvants as were thought desirable or necessary.

Operation. All patients were operated upon in the sitting position, using a special operating table.

Through a midline incision from the inion to the midcervical region a small bony opening is made in the occiput including the posterior rim of the foramen magnum, measuring in all some 4 cm. in diameter. A small linear opening is then made in the dura mater over the cisterna magna just to one side of the midline and the occipital sinus and on the side of the posterior parietal burr hole made for ventriculography. If the diagnosis is correct the cistern should be of large size and it is only rarely that a tonsillar herniation has been found. The arachnoid over the cistern should not be opened at this stage. The posterior fossa wound is covered with a wet pack and a subgaleal tunnel is made between the upper aspect of the wound and the previously made posterior parietal incision, this latter incision having been reopened. A No. 3 or No. 4 soft rubber catheter is passed through the parietal burr hole and into the lateral ventricle so that some 4–6 cm. will lie in the ventricle. The distal end of the catheter is passed into the posterior fossa wound via the subgaleal tunnel. It is unnecessary to make any side openings into the catheter inserted into the lateral ventricle.

Attention is now turned to the lower end of the catheter, which is cut so as to reach about 1 cm. below the lowest end of the dural opening over the cistern. The cut is made on the bevel so that the opening points inwards. A small opening is then made in the arachnoid over the cisterna magna and through this arachnoid opening the distal end of the tube is passed. The catheter is held in place with one black silk suture which holds the distal end close to the posterior aspect of the cistern. The dura mater is closed with interrupted black silk sutures to make a watertight closure

and is reinforced with Oxycel or Gelfoam to prevent leakage. The wound is then closed with multiple rows of interrupted silk sutures.

The patients are nursed in the sitting position and if all proceeds well no special aftertreatment is necessary. On occasions it may appear that the tube has become blocked and then it may be necessary to aspirate the tube through the skin in the occipital region to re-establish drainage. If such is done then it is probably wise to perform daily lumbar punctures to encourage the flow of cerebrospinal fluid through the tube. If cerebrospinal fluid leaks around the tube it may be necessary to aspirate the subgaleal space, and should leakage continue a temporary ventricular drain in the opposite lateral ventricle may tide the patient over the initial postoperative phase.

Small Variations in Technique in the 25 Patients. A full curved cerebellar incision was made in 4 patients in the belief that a posterior fossa tumour was present. After a negative exploration a Torkildsen's short circuit was performed as described above. The arch of the atlas was removed in 12 patients, the arches of the atlas and axis in 1, and the laminae of C1–3 in 1 patient. This last patient had a condition which appears from the operative description to be an Arnold-Chiari malformation. There appears to be no difference in the results with or without removal of the atlas. That tonsillar herniation was not the indication for removal of the arch of the atlas in all such cases is evidenced by the fact that in only 4 cases were the tonsils below normal and in 3 of these were described as herniated.

In 1 patient a circular opening was made in the occipital bone leaving the posterior rim of the foramen magnum intact. This patient died postoperatively.

In 1 instance a polythene catheter was used but proved very difficult to insert.

Postoperative Complications. There were 5 postoperative deaths. In 3 cases the cause of death was not established post mortem but the diagnosis of aqueduct stenosis was confirmed. One patient died on the third and 2 on the fifth postoperative days. One patient died from gastroenteritis 14 days after operation. The fifth patient died from a coliform meningitis and ventriculitis 6 weeks after operation. This was the only proven infection in the series although many patients showed evidence of meningeal irritation in the postoperative period presumably caused by presence of the rubber catheter.

In 2 patients a hemiplegia developed as a result of operation and in neither case have we adequate reason to explain it.

One patient required a revision of the Torkildsen's operation after 31 months and her result below is assessed from the time of the second operation.

LATE RESULTS OF OPERATION

One patient died after discharge from hospital and 7 months after operation from what seemed to have been the effects of a continued rise of intra-

cranial pressure (i.e., failure of operation). One patient was obviously deteriorating 3 months after operation.

Two patients were worse, physically, 14 and 39 months after operation although their intracranial pressure was restored to normal.

Three patients, 18, 19 and 32 months after operation, appear to have had their symptoms arrested, but, as regards vision and ability to work, are no better than before operation. These patients have been saved from a worse future.

Nine patients have done well and are at school or full work although one of them is blind. Two of these patients have been followed for $1\frac{1}{2}$ years, 1 for 2 years, 1 for $2\frac{1}{2}$ years, 2 for 3 years, 1 for $3\frac{1}{2}$ years, and 2 for 4 years.

Four patients operated upon 1, 2, 6 and 8 months ago are improving.

These results are summarised in Table 3.

TABLE 3

Results of ventriculocisternostomy

Died as a result of operation or consequences of the primary condition	6
Deteriorating as a result of failure of operation	1
Made worse by operation although the hydrocephalus was arrested (hemiplegia produced)	2
Total failures	9
Condition arrested by operation but patient not improved economically	3
Condition arrested and patient in full-time occupation	9
Too short a follow up although patient improving when last seen	4
Total successes	16

We thus have evidence that the short-circuit operation has proved satisfactory in 18 cases (73 per cent) although successful economic results were obtained in only 9 out of 21 followed for more than a year (43 per cent).

Failing vision is often a presenting symptom and it is of interest to investigate the changes in vision following a short-circuit operation.

In 12 cases the vision remained the same as at the time of the initial examination. In 4 instances there was improvement in vision, the actual changes being from 6/12 or 6/18 to 6/9 or better. No patient with a vision of less than 6/18 was improved.

In 3 patients there was a deterioration of vision after operation. One patient with 6/12, 6/18 vision before operation is now blind 47 months later, but he has no symptoms of raised intracranial pressure. Another with 1/60, 6/36 is almost blind 14 months later and a third with 6/12 and light perception now has 6/60 and nil 2 months after operation. All have secondary optic atrophy. The history of the last patient was of 3 years' duration and surely examination would have disclosed the papilloedema which must have been

present during most of this time. The history of the other 2 patients was brief and the papilloedema was severe on the first examination.

It appears from our results that although we are able to arrest hydrocephalus caused by aqueduct stenosis and so to relieve the symptoms of raised intracranial pressure, visual deterioration is unlikely to be reversed by a short-circuit operation and indeed may decline further with the appearance of secondary optic atrophy. The earlier the diagnosis can be made and treatment undertaken, the better will be the late results of surgery, for it has been shown that over three-quarters of the operations are successful so far as the short-circuiting procedure is concerned.

SUMMARY

A review of operative procedures and the results of operative treatment of patients with aqueduct stenosis is given.

The clinical picture of patients suffering from aqueduct stenosis is summarised in a series of 25 personal cases.

The ventriculographic appearances of the obstructed aqueduct are shown to be of 4 types.

The operation of ventriculocisternostomy as performed in this clinic is described, and the early and late results of this procedure are given for a consecutive series of 25 patients with non-neoplastic stenosis of the aqueduct. The operation is successful in nearly three-quarters of the patients.

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RESULTS OF DIRECT ATTACK ON NONFISTULOUS INTRACRANIAL ANEURYSM WITH REMARKS ON STATISTICS

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STATISTICS of successfully treated intracranial aneurysms are being more frequently recorded as larger series are developed. When these are broken down, various impressions are conveyed. This is particularly true when the lesions are individually described. Concerning such statistics, it seems misleading to speak in generalities. Aside from denominators common to all aneurysms, subarachnoid hemorrhage in particular, as well as intracerebral hematoma, "vasospasm," etc., many other factors come into play. We are speaking today of intracranial aneurysms as intracranial tumors were spoken of 40 years ago. As tumor differentiation is necessary for prognosis, so aneurysm differentiation is necessary depending upon type, anatomical location and the complications of the lesion.

In the hands of most surgeons, excluding Norlén and Olivecrona,[1] the aneurysms of the anterior communicating artery complex carry a much higher morbidity and mortality than do those on other parts of the circle of Willis. In our experience the pure carotid aneurysms, or those of the internal carotid-posterior communicating junction, offer the greatest chance of success and the anterior communicating aneurysms the least. There is also a perceptible difference when one compares the results of the surgical treatment of bleeding aneurysms or ones that have recently bled, with those that have never done so. These and many other factors make proper statistical orientation difficult.

It does not seem valid to discuss only successful treatment of aneurysms associated with subarachnoid hemorrhage operated upon 3 or 4 weeks following the initial hemorrhage and to exclude those that have been attacked earlier. Experience has shown that in the first 3 weeks of the disease, surgical therapy is more dangerous than when applied later, but in this time the patient is in the greatest danger of perhaps recurrent fatal hemorrhage. This then is the challenge in the surgical therapy of these lesions.

Seemingly significantly large series of successfully treated aneurysms become in reality very small when one considers the tremendous number of variables involved. Since we cannot compare properly unlike objects, we cannot compare, for example, aneurysms of the anterior communicating artery with those of the internal carotid artery. The problem of their treatment is usually entirely different. Ordinarily one thinks of 100 cases of a particular

lesion as a reasonably representative number. But if we break down 100 cases of aneurysms occurring in four or five common but different locations, the significant figure of aneurysms at a specific site drops precipitously, thus losing statistical value. The conditions under which these lesions are attacked must also be considered and include: rupture, the time of operation after rupture, the intracranial pressure at the time of operation, the presence of intracerebral hematoma, the exact location of the aneurysm, the technique employed (clipping, lobe resection, ligation, etc.), the physiological state of the patient's blood vessels and the neurological deficit. There should perhaps be a standardization of terminology. For proper evaluation of the effects of various forms of therapy the words "cure," "improved," and "satisfactory," should be defined. The follow-up period should be specifically stated. We should be clear in considering the results of operation. Survival rates should perhaps come first, followed by residual neurological deficit, rehabilitation, etc. The conditions seem endless. These are but a few of the important questions that must be answered before we can properly evaluate the results of surgical treatment of these lesions. There is little value in comparing the results of treating 2 aneurysms of the posterior communicating artery with 15 or 20 aneurysms of the anterior cerebral complex. This tells us only that in the particular series the latter lesion is more common. It seems then, until much larger series have been developed, that statistical records will not carry much weight. It will mean, for example, that 100 aneurysms of each specific arterial trunk will have to be reported, operated upon in the same clinic, by one or two closely associated surgeons, employing the same techniques, under the same conditions. With so many variables concerned, this seems at present an insurmountable task. The answer, it appears, lies in part in a greater, wider experience covering many more years. Accumulated active experiences should be continually recorded, for it is only by this means that our knowledge of specific aneurysms variously treated can help us determine the best method of management. Lastly, there is a distinct necessity for statistics on the mortality of each aneurysm problem when untreated. Certainly the mortality of treatment should not be greater than the mortality rate of the disease. From reports of relatively small series of surgically treated aneurysms, only a few conclusions can be drawn. This will depend upon what the reader is looking for as well as what the writer is trying to say.

In this report the results of direct surgical attack in 55 cases of intracranial aneurysms are presented. These do not compare favorably with those in some published reports. We are disappointed with our figures but feel that the conditions are not comparable. It is our impression that this is because of a number of factors unrelated to surgical technique. Our statistics include all patients operated upon. The operative procedures were accomplished as soon as possible after diagnostic methods were completed. Perhaps this is poor judgment. Our aim was to reduce the mortality and possibility of early recurrent hemorrhage most common in the second and third week after the

initial insult. The interval between the initial hemorrhage and operation varied considerably depending upon when the patient was referred to us following the bleeding episode.

MORTALITY

In our series, 55 patients with intracranial aneurysms were operated upon by the direct attack. Of these, 3 had multiple lesions. There was an over-all mortality of 45 per cent—25 of the 55 patients dying.

INCIDENCE OF SUBARACHNOID HEMORRHAGE

Of the 55 patients, 43 or 78 per cent, had had one or more subarachnoid hemorrhages. Of these 43, 21 patients died. Of the 12 patients without hemorrhage, 4 died. It is apparent that with a comparable number of cases the mortality rate of patients without hemorrhage would still be 28 per cent less than that of patients with hemorrhage.

INCIDENCE OF INTRACEREBRAL HEMATOMA

Intracerebral hematomas occurred in 16 patients, 14 of whom died. This is a significant fact, not mentioned by others. It is important to note also that 14 of these hematomas occurred in association with the anterior cerebral-anterior communicating complex and middle cerebral artery aneurysms.

MORTALITY AND MORBIDITY—PREOPERATIVE DEFICIT

It is impractical in a discussion of this type to consider all the preoperative neurological defects. What we have felt to be the most important in evaluation of the mortality and morbidity rate are noted. When operated upon, 11 per cent of the patients were in coma, 31 per cent had hemiplegia, and 42 per cent had cranial nerve disturbance (this last included those with hemiplegia or hemianopic defects because both were present in the same patient).

TIME OF OPERATION FOLLOWING ACUTE EPISODE

We considered next the time of operation and divided the patients into those who had "early" and "delayed" operation, taking 14 days as the dividing point. Of the 35 patients with "delayed" operations, 10 died. Of the 17 with "early" operations 13 died. The borderline cases, in which operation was performed on the 14th day, comprised 3 patients, all of whom died. The number of "delayed" cases was twice that of the "early." However with an equal number of cases the mortality rate would still be 30 per cent lower in those operated upon at a later period.

ANATOMIC LOCATION

As regards the anatomical location of the aneurysms, these were divided into six sites: 5 were located on the infraclinoid portion of the internal carotid artery; 25 on the supraclinoid portion of the internal carotid artery; 6

on the middle cerebral artery; 7 on the anterior cerebral; 10 on the anterior communicating and 2 on the vertebral arteries.

TECHNIQUE OF OPERATION

The technique employed in the treatment of the various lesions was as follows (in all situations where a hematoma was present, it was removed transcortically):

Infraclinoid Carotid Aneurysms. There were 5 patients with infraclinoid internal carotid artery aneurysms. Three of these were treated by intracranial clipping and extracranial carotid ligation, and 1 by extracranial carotid ligation and muscle packing. One patient had bilateral aneurysms and exploration only was made, the lesions being left untreated. Of these 5 patients, the latter 2 (40 per cent) died.

Supraclinoid Carotid Aneurysms. There were 25 aneurysms on the supraclinoid portion of the internal carotid artery. Of these, 8 were treated by trapping procedures through clipping of the internal carotid artery and extracranial carotid ligation. None of the patients so treated died. One of these aneurysms was reinforced by wrapping with cottonoid. Three had clips applied to the internal carotid artery on either side of the neck of the aneurysm.

Four aneurysms were trapped by clipping the internal carotid artery alone, on either side of the neck of the aneurysm. Two of these were reinforced with cotton. All 4 patients treated in this way recovered.

Ten aneurysms were treated by clipping of the aneurysmal neck, and 2 were reinforced with muscle or cotton. Three of these patients died.

There were 2 lesions at the bifurcation of the internal carotid artery. One was closed by ligation of the aneurysmal neck, the second by clipping and muscle wrapping. In this latter case simultaneous occlusion of the internal carotid artery in the neck was made with a Blalock clamp. At the time of dural closure with the clamp removed, a secondary hemorrhage occurred. The lesion was re-clipped and the muscle wrap removed. At autopsy the clips were found to be properly applied and all vessels were free of clot. There was softening of the ventral nucleus of the thalamus. The thalamoperforating branches of the middle cerebral artery had been occluded by clips.

Two patients who had hematomas associated with the aneurysm expired. One of these patients had multiple aneurysms. In this particular situation the problem was an acute one of hematoma evacuation and the aneurysm was not treated. Five (20 per cent) of the patients in this group died.

Middle Cerebral Aneurysms. There were 6 patients with middle cerebral artery aneurysms, 5 of whom had associated intracerebral hematomas. There were 4 deaths (66 per cent) in this group. In 3 cases the hematoma only was removed and the aneurysm was untreated. All 3 of these patients died. In 1 case the neck of the aneurysm was clipped following the removal of a

hematoma. The patient with this lesion died. Of the remaining 2 cases, in 1 with aneurysm and hematoma, the hematoma was evacuated, and the neck of the aneurysm was clipped and reinforced. In the other the lesion was reinforced only.

Anterior Cerebral Aneurysms. There were 7 anterior cerebral aneurysms in this series, 3 of which were associated with hematomas. All 3 patients with this combination died. One aneurysm was trapped; the patient with this lesion expired. The aneurysmal neck was clipped in 2 patients, in one of which a reinforcement procedure was accomplished. One patient had clips applied to both anterior cerebral arteries and 3 had clipping of the anterior cerebral artery proximal to the aneurysm. Only 1 of these last 3 patients recovered. In this group of 7 cases, 6 (86 per cent) of the patients died.

Anterior Communicating Artery Aneurysms. The anterior communicating aneurysms comprised 10 of the total series. Six of these were associated with intracerebral hematomas. The 6 patients with aneurysms and hematomas all died. Of the 6 with hematomas, clots were removed in all. A muscle pack with clipping of the aneurysm was accomplished in 1, and muscle packing with extracranial ligation of the internal carotid artery was done in another. In a third, after the clot was removed, the anterior cerebral artery and the anterior communicating artery were clipped and a muscle pack was placed. In the fourth the aneurysm was untreated. In the fifth, intracranial internal carotid artery clipping was made. Extracranial internal carotid artery ligation was done in the sixth.

In 3 of the cases of anterior communicating aneurysm clips were applied to both anterior cerebral arteries and the aneurysm was removed in 1. One of these patients recovered but was severely deteriorated and has remained so.

In 1 patient the neck of the aneurysm was clipped and reinforcement with muscle was accomplished. This patient recovered.

In this group of anterior communicating aneurysms 8 (80 per cent) of the patients died.

Vertebral Artery Aneurysms. The 2 vertebral artery aneurysms were found in patients both over the age of 65 years. One had had typical trigeminal pain over the first division. The trigeminal nerve was approached by the posterior route and a vertebral artery aneurysm discovered. Treatment was by nerve root section. The other patient had cervical and suboccipital pain and dizziness. Operation disclosed a large vertebral artery aneurysm of calcific nature around which were entwined the rootlets of the 10th and 11th and upper cervical nerves. An external neurolysis of the nerve rootlets from the aneurysmal mass was accomplished. Both of these patients were relieved. The second patient had only one vertebral artery.

RESULTS OF OPERATION

Of the 55 patients operated upon, 25 expired and 25 were described as improved. In 2 patients there was an increase in the neurological deficit. The

remaining 3 patients were unimproved; in these there was no change in the neurological deficit and no evidence of recurrent hemorrhage. Of these 5 patients 3 had supraclinoid carotid artery aneurysms. Two were trapped intracranially, and 1 aneurysm was removed. In the latter case, the patient had a preoperative hemianopsia. Following operation the visual defect cleared but the patient has a permanent hemiplegia. In a fourth patient with a complete external ophthalmoplegia, 3 days following intracranial trapping of the aneurysm, a hemiparesis developed which receded practically completely. In the fifth case the patient had an anterior communicating artery lesion, and both anterior cerebral arteries were clipped. A preoperative hemiplegia regressed but there developed a progressive deterioration with convulsive activity. Three of the patients were operated upon in the "late" and 1 in the "early" period following subarachnoid hemorrhage. The other patient did not bleed.

POSTOPERATIVE FOLLOW-UP

Of the 29 patients who survived operation, a follow-up report was obtained on 28. Two patients had died—1 of "peritoneal sarcoma" 7 years following surgery for an infraclinoid aneurysm, and 1 of a perforated peptic ulcer 4 months after operation for a supraclinoid lesion. The first patient had led a normal life until the time of her death. The other had a preoperative hemiparesis from which she did not recover.

Of the remaining 26 patients, 23 were re-examined. The other 3 could not appear because of distance. Their progress was reported by correspondence.

The longest follow-up period was 10 years, the shortest 5 months, with an average of 7.9 years.

In the group of patients still alive at the time of the last report, there were 18 supraclinoid aneurysms, 2 infraclinoid, 1 anterior cerebral, 2 anterior communicating, 2 middle cerebral, and 2 vertebral aneurysms.

Of the 18 patients with supraclinoid carotid artery aneurysms, 15 were doing the work they had done before operation, 1 was totally disabled, 1 was working with limitation and attending college, and 1 with a preoperative bitemporal hemianopsia had a postoperative hemiparesis with return of full fields of vision. All patients in this group with extraocular nerve disturbance showed improvement in this deficit though incomplete. This group comprised 8 patients. The vision in 2 patients with hemianopic defects had improved. There were 2 patients with preoperative hemiparesis. One has recovered completely; the other, though ambulatory, requires nursing care.

The 2 surviving patients with infraclinoid aneurysms were doing their usual work, 9 years and 10 years respectively following operation. One had a residual fixed pupil and optic atrophy. The other had no neurological deficit.

The 1 surviving patient with an anterior cerebral aneurysm is perfectly well and performing the duties of housewife and mother.

The 2 patients with anterior communicating aneurysm are hopeless

neurological cripples, both showing profound frontal lobe disturbance. One of these is the patient on whom a recent follow-up report could not be obtained. He was last heard from 2 or 3 years following operation and had not improved.

Of the 2 patients with a middle cerebral artery aneurysm, the preoperative hemiplegia and hemihypalgesia of 1 had not receded. The other patient still is having her preoperative convulsive activity but is doing useful housework.

One of the 2 patients with a vertebral aneurysm has never returned to work because of continued disturbance in balance. The other is symptom free.

DISCUSSION

It is difficult to interpret these figures for the reasons given above. However, several conclusions can be drawn. First, that intracranial aneurysms associated with intracerebral hematoma carry a very high mortality. The presence of a hematoma implies early operation, to remove an expanding mass which is endangering the patient's life. In our hands this has resulted almost invariably in fatality. Of 16 patients with intracerebral hematoma, 14 (87 per cent) died. These hematomas were associated with anterior communicating-anterior cerebral complex aneurysms in 9 instances, and with aneurysms of the middle cerebral artery in 5—sites that in this series have shown the highest mortality rate. Concerning the initial neurological deficit, one notes also that among patients with hematoma, 4 were in coma, 4 had papilledema and 9 were hemiplegic. Of 6 patients in coma, 4 died, and of the 17 patients with hemiplegia, 10 died. These features, then, were important factors in the mortality figure. The type of operative procedure and the age of the patient had little effect on the statistical mortality.

From our experience we draw the same conclusions reached by Norlén and Olivecrona,[1] i.e., that operation for intracranial aneurysm should be deferred, if possible, 3 weeks following the subarachnoid hemorrhage. The mortality of operation within this period is as great as when the disease is allowed to run its natural course over the same time interval. There is little question that early operation is dangerous but the hazard is caused not solely by early surgery when "vasospasm" is felt to be a most important factor, but in great measure by the profound neurological deficit which may or may not be incident to an expanding destructive hematoma. In cases of supraclinoid lesions, in which the incidence of hematoma is very low, the incidence of recovery is high. A high percentage of patients will die in the early period following a subarachnoid hemorrhage with or without surgery. Present techniques (carotid ligation, the use of sympathetic blocking agents to combat vasoconstriction, and hypotensive drugs) offer little assurance that the patient can be protected from a recurrent, perhaps fatal hemorrhage before the optimal time for surgery has been reached. Until more certain

methods are developed, the alternatives are a high mortality without surgery or a chance at survival with surgery though admittedly this, too, is prohibitive.

ADDENDUM

Since June 1954, 3 aneurysms have been attacked intracranially: 1 was supraclinoid in location, 1 infraclinoid and 1 was situated at the carotid bifurcation. The first 2 patients have recovered without defects. The third is hemiplegic.

REFERENCE

1. Norlén, G., and Olivecrona, H. The treatment of aneurysms of the circle of Willis. *J. Neurosurg.*, 1953, *10:* 404–415.

CEREBRAL HEMISPHERECTOMY FOR CONTROL OF INTRACTABLE CONVULSIVE SEIZURES*

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THIS is a report of our experiences with cerebral hemispherectomy for the control of intractable convulsive seizures. It is a direct outgrowth of a former project of performing hemispherectomies in an attempt to cure patients with cerebral gliomas. The first patient operated upon primarily for seizures was a 38-year-old man who in February, 1950, had a left cerebral hemispherectomy in an attempt to control persistent status epilepticus. He had been in status intermittently for a period of 6 weeks, controlled only when under sodium pentothal anesthesia. Twelve years previously, this patient had undergone removal of a left parietal glioma. It was felt that his seizures were caused by either recurrence of the tumor or extensive glial scarring secondary to the removal of the tumor. Following removal of the entire left hemisphere in 1950 he has had no seizures. He does have a persistent right hemiparesis but is able to be up and around and can do minor jobs around the house. Before hemispherectomy he had a global type aphasia but now he has fair speech comprehension and production. Subsequently 8 more patients have been treated by a similar operative procedure. All have been severe uncontrolled epileptics who have had a hemiparesis from infancy or early childhood. The purpose of this paper is to present the results of cerebral hemispherectomy on these 9 patients. The discussion will be limited primarily to its effect on the control of seizures and to the pre- and postoperative neurological and mental status.

REVIEW OF THE LITERATURE

Total, unilateral cerebral hemispherectomy was first reported by Dandy[5] in 1928. In all his 5 cases the operation was done in an attempt to cure gliomas. Others[1,4,6,7,14,15,17,18,21] have since reported on hemispherectomy for attempted cure of gliomas. Williams and Scott[20] stated that McKenzie in 1938 had done a cerebral hemispherectomy in an attempt to control convulsive seizures. There were no further reports of the procedure being done for this purpose until November, 1950, when Krynauw[8,9] described a series of 12 patients upon whom he had done hemispherectomies. All of these patients had infantile hemiplegias associated with uncontrolled convulsions.

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Since Krynauw's original paper, others[3,10–13,16] have reported limited experience with this procedure.

SELECTION OF CASES AND PREOPERATIVE STUDIES

Included in this series from the University of Minnesota is the above-mentioned patient plus 8 more patients who had an infantile type of hemiplegia associated with uncontrollable seizures. All the latter were institutionalized epileptics from the State School and Hospital at Cambridge, Minnesota. The main criteria used in the selection of these patients were that they must have had a hemiparesis since infancy or early childhood and also grand mal seizures uncontrolled by what is generally considered adequate anticonvulsant medication. Hemiparesis alone was never considered sufficient indication for the operation. Krynauw,[8,9] however, operated on several patients who had no seizures but who did have infantile hemiplegias associated with severe personality disturbances which prevented them from living a normal life in society. The age of the patients in our series varied from 13 to 38 years and at the time of operation they had been institutionalized for periods varying from 6 to 17 years. Neither the age of the patient nor the length of time hospitalized prior to surgery had any effect on the postoperative control of the seizures. The long institutional care of these patients did make available very important data, i.e. the number of observed seizures within each 24-hour period for many years and the kind and quantity of anticonvulsant drugs given during these periods. This type of information is not commonly available in a study of seizures because these data usually are obtained from the patient, a relative, or a friend, and represent a rough approximation of what actually exists and may be grossly misleading. All patients were examined carefully to ascertain their preoperative neurological status. Other preoperative studies done on all patients were electroencephalograms, pneumoencephalograms, carotid angiograms, and an extensive battery of psychological tests.

Preoperatively there was evidence of gross abnormality of the affected hemisphere as manifested not only by the hemiplegias and the seizures but also by the above-mentioned studies. Air encephalograms usually showed a dilated ventricle on the affected side indicative of an atrophic process or, as in several cases, large porencephalic cysts. Carotid angiograms often revealed malformation or atresia of the middle cerebral vessels.

OPERATIVE PROCEDURE AND FINDINGS

All patients were operated upon under pentothal-curare-nitrous oxide-oxygen anesthesia. A large craniotomy flap was turned to expose a maximum of the cerebral cortex. After the dura mater was opened the cortex was inspected with a Wood's lamp, the patient previously having received 5 cc. of 20 per cent sodium fluorescein. Cortical electrography was carried out under various experimental conditions. Arterial and venous blood samples were taken for oxygen-carbon dioxide saturations.[19] Ligation of various

vessels was done to determine the effect on the cortical electrogram. The hemisphere was then excised. The middle cerebral artery was ligated and cut approximately $1\frac{1}{2}$ cm. distal to its origin and if the posterior cerebral artery were readily visualized at this time it was clipped and cut at its origin from the posterior communicating. If it could not be visualized easily at this time it was exposed and cut later, just prior to the final removal of the hemisphere. A linear incision was made through the corpus callosum and the lateral ventricle was entered. The anterior cerebral artery was cut just distal to the anterior cummunicating branch but only after ascertaining that the contralateral anterior cerebral vessel was present and arising from the contralateral carotid artery. The stria terminalis was then visualized. An incision was made along the stria so that the caudate nucleus was separated from the thalamus. The line of dissection then was carried down the internal capsule to emerge at the incisura tentorii. It was during this latter dissection that the posterior cerebral artery sometimes was cut. The choroid plexus was excised.

Electrograms were taken from the basal ganglia and from the exposed parts of the remaining hemisphere. The cavity was filled with saline solution and closure was obtained in the usual fashion.

This type of resection removes all structures peripheral to the thalamus. Great care was taken to insure the removal of the hippocampus and the orbital gyri which may get tucked underneath a fold of arachnoid and be missed.

The extent of the hemispherectomy varied slightly from case to case

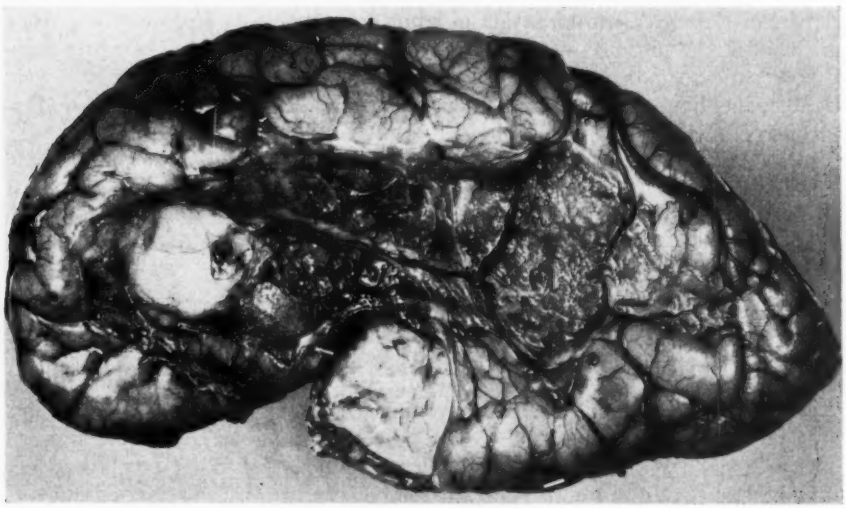

FIG. 1. Photograph of excised hemisphere from patient with seizures and hemiplegia. There is extensive scar formation in cortex and an overlying subarachnoid cyst.

FIG. 2. Photograph taken at time of operation. The thickened pia-arachnoidal membrane is visible. The underlying cortex was adherent to this membrane. This hemisphere was excised.

but in general the entire cerebral cortex including the insula, the hippocampal gyrus, and the inferior medial orbital gyri were removed. In 2 of the earlier cases, the dissection varied so that the caudate nucleus was left intact.

The gross abnormalities seen at operation were very extensive in all cases and included such diverse abnormalities as huge areas of severe leptomeningeal scarring, subarachnoid cysts that had replaced large areas of the brain, and extensive vascular malformations (Figs. 1 and 2). None of the hemispheres removed was normal on gross or microscopic examination.

In general, the preoperative EEGs showed a severe, diffuse dysrhythmia that was more pronounced on the affected side. Postoperatively there was routinely a dramatic improvement in the appearance of the EEGs. The rhythms from the remaining hemisphere became normal or borderline normal patterns. The side of the head from which the hemisphere was removed revealed a rhythm similar to the normal (remaining) side except for a decreased voltage (Figs. 3 and 4).

REPRESENTATIVE CASE REPORT

History. R. P. was born Oct. 6, 1917. At the time of birth a left hemiparesis was noted. Otherwise she developed fairly normally until the age of 2 at which time she fell from a couch and struck her head. She began to have grand mal type convulsive seizures. She continued to have intermittent attacks throughout infancy and child-

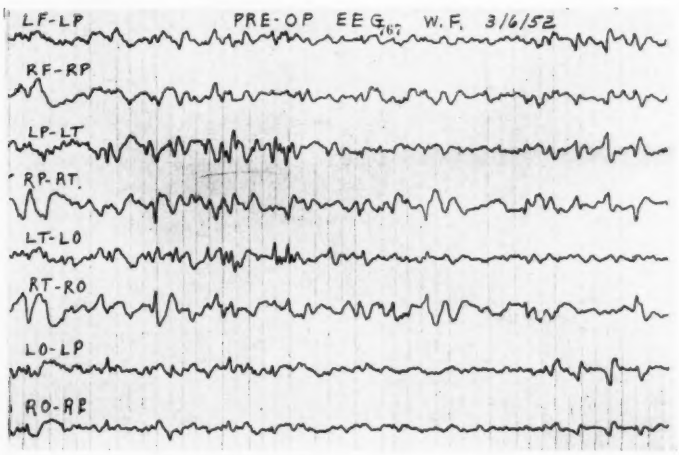

FIG. 3. Preoperative electroencephalogram revealing diffuse dysrhythmia bilaterally. The right hemisphere was eventually excised.

hood. In 1937, at the age of 20, her seizures became much more frequent and severe. Because of grand mal attacks and an increasingly severe personality disturbance, it became very difficult to care for the patient at home. She was studied at the University of Minnesota Hospital in 1947 and commitment to the State School and Hospital at Cambridge was advised. She entered there several months later. Following admission to Cambridge, she complained of numerous psychosomatic symptoms and her seizures continued. These seizures were of two types: (1) minor spells described by the patient as "blackout spells." These attacks were preceded by a feeling of pressure in the back of her head, dizziness, and a griping sensation in her abdomen.

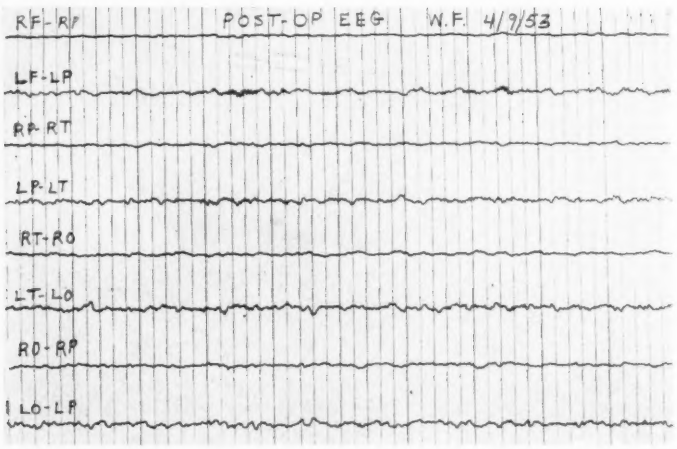

FIG. 4. Electroencephalogram made 6 months after right cerebral hemispherectomy. There is some amplitude asymmetry but the fundamental rhythm is that of the left, unoperated, side.

They lasted from a few seconds to 5 minutes and occurred five to ten times per day. (2) Over a period of years, the above attacks were followed about once weekly by a typical grand mal convulsion, severe in type and duration.

Anticonvulsant medication did not appreciably affect the attacks. Because of her hemiparesis and uncontrolled seizures, she was selected as a candidate for hemispherectomy.

Preoperative Examination. She was an obese 36-year-old woman who was reasonably alert and who was fully oriented and cooperative. On March 10, 1953, a Wechsler-Bellevue I.Q. showed a verbal scale of 78 and she obtained a score of 70 on the Stanford-Binet Form L. Positive neurological findings were a left homonymous hemianopsia, a left 6th nerve palsy, and bilateral horizontal nystagmus with a slight rotary component. There was a definite left hyperreflexia with a positive Babinski sign and she had a moderately severe spastic left hemiparesis with the arm being most involved. She walked with a typical left hemiplegic gait. Her entire left side was moderately atrophied, the upper extremity more than the lower. There was a severe loss of all sensory modalities on the left side.

FIG. 5. Pneumoencephalogram made prior to excision of the right cerebral hemisphere. There is considerable shift of the ventricular system to the right and the right ventricle is dilated.

Roentgenograms of the skull revealed a slight asymmetry—the right side being somewhat underdeveloped. A pneumoencephalogram revealed the right ventricle to be greatly dilated and the entire ventricular system was displaced to the right (Fig. 5). Preoperative EEG showed random slow waves without a definite basic frequency. The voltages over the left side were higher than on the right.

Operation. On March 23, 1953, the right cerebral hemisphere was removed. This included the insula, the hippocampal gyrus, and the caudate nucleus.

Postoperative Course. A half hour after completion of the operation, she was alert and rational. She could move her left arm and leg almost as well as preoperatively and she could identify well pain and light touch stimuli even though this ability was definitely disturbed. The left arm and leg were flaccid. There was a definite left hyperreflexia, with a very positive left Babinski sign.

When seen 1 month later, she was walking as well as preoperatively. She had had no seizures of any type. She was on no anticonvulsant medication. An EEG at that time revealed an amplitude asymmetry between the two sides with less activity over the posterior half on the right—that is, the side operated upon. The activity from the left parieto-occipital area was normal alpha activity.

She was last seen 17 months postoperatively. At that time her gait and reflexes were the same as prior to surgery. She had some finger movement in the left hand at least as good as the preoperative level. The nystagmus present prior to surgery

was gone. Her Wechsler-Bellevue verbal scale was 82 and the Stanford-Binet Form L was 74—both slightly higher than preoperatively. She had had one very brief akinetic type seizure so she was placed on mebaroin therapy. Both the patient and her mother have been more than satisfied with the result of her operation.

A summary of the 9 cases is given in Table 1.

TABLE 1

Summary of patients with infantile type hemiplegia and intractable convulsive seizures who had cerebral hemispherectomy prior to April, 1954

Patient	Sex	Age	Diagnosis	Followup in Months	Preop. Seizures per Year	Postop. Seizures	Post op. Medication
H.P.	M	38	Status epilepticus	57	Status epilepticus	0	none
K.W.F.	M	13	Birth injury	25	165 G.M.	0	none
C.K.	F	13	Sturge-Weber	29	300–500 G.M.	1	yes
O.L.	F	34	Encephalitis	29	300–500 G.M.	5	none
C.B.	F	19	Microcephaly; encephalitis	13¼ (died)	700–800 G.M.	80	yes
R.P.	F	35	Birth injury	21	50 G.M. 1800–3600 P.M.	1	yes
D.S.	M	30	Birth injury	19	50–100 G.M.	1	yes
D.G.	M	15	Birth injury	12	65–160 G.M.	0	none
E.B.	F	28	Encephalitis	7	200–300 G.M.	0	none

EFFECT ON CONTROL OF SEIZURES

All patients except the previously mentioned man with a glioma had had seizures since infancy or childhood. With the exception of this man, all others had uncontrollable seizures that made institutionalization necessary. The postoperative followup now ranges from 7 to 57 months. Of the 9 patients, 8 have had excellent results so far. Four of the patients have had no seizures of any kind and are on no medication. Three others have had one seizure each—all occurring in the first few months postoperatively. They are now on small doses of anticonvulsant medication. One patient had five seizures in the immediate postoperative period but none in the last 2 years and now is not on medication. One case is classified as a poor result although the reduction in seizures was almost 90 per cent. In retrospect this patient with a definite microcephaly and a history of having had a severe meningo-encephalitis at the age of 5 weeks was probably poorly chosen. She had averaged 700 to 800 grand mal seizures each year for a period of almost 17 years of institutional custody. She lived 13¼ months postoperatively and then died of diphtheria in the state institution. Prior to her death she had about 80 grand mal attacks.

EFFECT ON MOTOR SYSTEM

Eight of the 9 patients in this series had become hemiplegic in infancy or early childhood, and while able to walk, all had a moderate to severe spastic hemiparesis most severe in the upper extremity. For the most part,

the affected arm was used very little except for gross movement at the shoulder and elbow joints. In all the patients, however, movement at these joints was very strong. Four of the patients had some degree of use of their fingers and in 2 this finger movement was adequate to be used in dressing themselves, picking up small objects, etc. There was a moderate to severe degree of atrophy on the affected side in all the patients. The deep tendon reflexes were extremely hyperactive with knee and ankle clonus usually present. The Babinski response was invariably positive.

Within a half hour to several hours following completion of the operation, all patients became sufficiently responsive to carry out adequate testing. In most of the patients at this time, movement was present in both the arm and leg. The previously spastic extremities were now flaccid but the reflexes and toe signs remained the same as preoperatively. Within a week after operation 2 patients could walk unassisted and the other 7 did so within 10 to 30 days. The earliest return of walking ability came on the third postoperative day. When examined several months later, all had regained their preoperative walking status and in 1 patient this ability was considerably improved. With lessening of the spasticity of the extremities, unstable joints had increased range of movement which made orthopedic deformities more apparent.

Ability to use the affected arm and fingers also returned to the preoperative level but this function was regained more slowly. Finger movement was not abolished by the procedure. Whatever finger function existed prior to surgery was soon regained. One patient volunteered the information that he could now use his fingers and arm without purposely thinking about it such as was necessary preoperatively. One patient with a Sturge-Weber syndrome and a severe spastic hemiparesis, had marked athetoid movements of the involved arm and almost continuous bobbing of the head. Both these signs disappeared completely following hemispherectomy.

It has long been accepted that abdominal reflexes are abolished by contralateral lesions of the premotor cortex, although it has been pointed out by Bucy[2] that this is not necessarily true. He found that removal of the premotor cortex may temporarily reduce or abolish the contralateral abdominal reflexes, but subsequently they return. Several of the patients in this series had perfectly normal, brisk abdominal reflexes bilaterally following hemispherectomy.

The effects of removal of the entire cerebral cortex as well as most of the basal ganglia might be expected to exaggerate a pre-existing hemiparesis. In no case did this occur. Krynauw[8,9] stated that the integrity of the caudate nucleus on the side opposite the hemiparesis was necessary for the maintenance of preoperative motor function. This in our experience is not true since 5 of our patients with complete removal of the caudate nucleus recovered motor function to the same extent that others did with the caudate nucleus intact. It is felt that the rapid return of motor function to the preoperative level is because of the contralateral hemisphere having assumed many years previously the function of the pathological side. Possibly the

pathological hemisphere serves only to exert a restraining effect on the normal side. This restraint appears to act in similar manner in the control of seizures, in the appreciation of somesthetic sensation and in the disorganization of personality and intellect.

EFFECT ON SENSORY SYSTEM

It must be appreciated that the various modalities of sensation are extremely difficult to evaluate in patients of the type under discussion. Eight of the 9 patients were institutionalized epileptics and were mentally deficient in varying degrees preoperatively. Intelligent cooperation in sensory testing was not always present. Most of the patients preoperatively had some evidence of sensory deficit in all modalities. From postoperative testing in those patients who were sufficiently intelligent to cooperate, the following generalizations can be made: neither superficial or deep sensation is abolished by hemispherectomy but a quantitative change is apparent. It is felt that this is caused by a loss or reduction of the finer discriminative powers and not by the abolition of either gross superficial or deep sensation. The most adequate testing that could be carried out was done on a 20-year-old boy with a Wechsler-Bellevue I. Q. of 86. Prior to surgery he was the only patient who had grossly normal sensation in all modalities. In the first few days postoperatively all modalities were still present but were now less well delineated than prior to surgery. His responses at this time were quite accurate but delayed. When seen several months later there was essentially no difference in sensory perception between his two sides. He could accurately identify small objects placed in his fingers, his ability to perceive figure writing on his skin was normal, and position and vibration senses were intact. There was no disturbance in recognizing various parts of his body. It has previously been accepted that stereognostic sensation was appreciated only in the contralateral parietal cortex. That this is not entirely true is shown by this patient's ability to identify deep sensation without his contralateral parietal cortex. It must be assumed that either the parietal cortex is bilaterally represented or that the thalamus also can act in the appreciation of this type of sensation.

EFFECT ON SPEECH

Six of the 8 patients had left hemiplegias and for practical purposes had used only their right arms for many years. None of these patients had any loss or change in speech and all could talk as soon as they had recovered from anesthesia. Of the 3 patients who had right hemiplegias, 1 had had a severe global type aphasia following operation for a left-sided brain tumor 12 years previously. Following hemispherectomy he could make simple wants known, and had a fair degree of comprehension. The other 2 right hemiplegic patients had normal speech preoperatively and this was unchanged by surgery. It would seem that the hemisphere removed is always the nondominant one since these patients suffered their loss of function many years previously. It is apparent that handedness in patients with infantile type hemiplegia has no relation to speech function and that handed-

ness is always transferred to the opposite side following the onset of paralysis. This, of course, agrees with previous concepts for it has long been known that transference of speech function is the rule rather than the exception in children up to about the age of 15.[4]

EFFECT ON INTELLECT AND PERSONALITY

An extensive battery of pre- and postoperative psychological tests were given to 8 of the 9 patients. The results in 7 of these cases have been reported elsewhere.[18] No loss of intellectual function as measured by the various testing procedures was found. In fact, most of the patients scored slightly higher postoperatively. It is quite possible that this improvement might be caused by release from the effect of large doses of anticonvulsant medication. Another factor could be release of the relatively normal hemisphere by what Krynauw[8] referred to as "the abnormal driving of the pathologic side." This is dramatically shown by the change in the EEG pattern. The clinical impression expressed by a group of physicans and psychiatric aides, as well as the clinical psychologist's findings, indicate that these patients as a whole are a definitely happier, better adjusted group than they were preoperatively. Striking general improvement was noted in a girl 13 years old who preoperatively could not feed herself, was neither bladder nor bowel trained, and who sat around the institution with a dull and lifeless appearing face. She was also a very difficult behavior problem. The child is now feeding herself, taking care of her toilet needs, and is no longer subject to violent temper tantrums. Her change in behavior and appearance could hardly be more complete.

As stated previously, Krynauw[8] operated on several patients strictly on the basis of a severe behavior disturbance associated with an infantile hemiplegia. The patients had no seizures. Whereas patients in the University of Minnesota series were not operated upon for behavior disturbance per se, several were markedly benefited in this aspect of personality adjustment. For example, 1 of the patients had been particularly difficult to manage prior to surgery; it was frequently necessary for her to be placed in seclusion to protect others and herself from her violent tantrums. Postoperatively she became cheerful and cooperative and the previous flareups have not been manifested. Since such behavior disturbance itself is even more incapacitating socially than are seizures, it is felt that severely disturbed patients with infantile hemiplegia and with only a few or no seizures might well be considered as candidates for hemispherectomy.

SUMMARY

There have been no operative deaths in this series. One patient died of diphtheria 13¼ months postoperatively. The results in these 9 cases of cerebral hemispherectomy for intractable seizures have been very gratifying. Eight of the 9 patients have been for practical purposes completely relieved of their seizures for periods of 6 to 57 months. The remaining patient had a 90 per cent reduction of seizures up to her death 13¼ months postoperatively.

Most of the patients are now taking no anticonvulsant drugs. The pre-existing neurological deficits have not been aggravated and in at least one there has been a definite improvement. According to psychometric evaluation no patient has had his intellect reduced and several have shown a definite improvement in their personality adjustment. The patients and their relatives are very gratified by the results and further patients are going to be operated on.

This study has been made possible through the cooperation of Dr. Raymond Gully, Superintendent of the State School and Hospital, Cambridge, Minnesota. Mr. Jarle Lierfallom, Director of Public Institutions, State of Minnesota, made available funds providing for these patients' care. The above study is to be continued under the auspices of the U. S. Public Health Service, Council on Neurological Diseases and Blindness.

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OBSERVATIONS ON VENTRICULAR AND LUMBAR SUBARACHNOID PERITONEAL SHUNTS IN HYDROCEPHALUS IN INFANTS*

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THE treatment of progressive hydrocephalus has challenged for decades the ingenuity of general surgeons and, also, neurosurgeons since the infancy of our specialty.

Cushing's remarks in 1926 are to the point: "among the first of my patients—as will probably have been true of most young men entering this particular field—were infants with what is known as essential hydrocephalus, for which a greater number of treatments have as yet been advocated (I have been guilty of proposing one or two myself) than successes recorded—if indeed there are any clear-cut successes recorded."[2]

We also plead guilty, not of excessive youth, but of early enthusiasm in approaching this problem and would like to present our trials and tribulations with this baffling condition. We hope that our successful cases will stimulate others to further interest and our failures offer data that will save others some of our headaches.

This presentation is primarily concerned with the ventricular and lumbar subarachnoid to peritoneal cavity shunts in infants having progressive hydrocephalus (Fig. 1).

We have done 10 ventricle to peritoneal cavity shunts in children and adults with obstructive ventricular lesions or tumors which did not respond to the Torkildsen procedure. Although the results were grati-

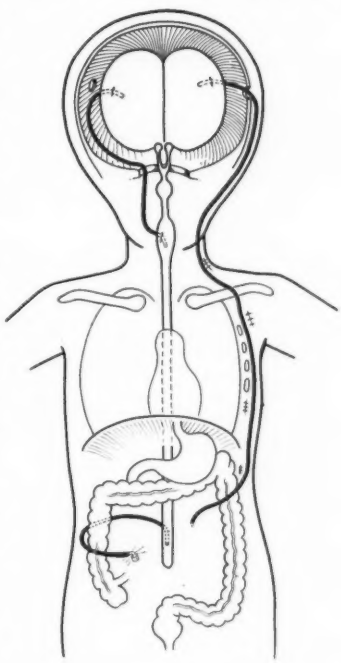

FIG. 1. Peritoneal shunts used for primary operation (see text).

* Presented before The Harvey Cushing Society, Santa Fe, New Mexico, May 7, 1954 and the Surgical Forum, Clinical Congress of the American College of Surgeons, Atlantic City, November 19, 1954.

fying in some, the short followup does not justify any conclusions at this time.*

One might challenge our judgment in operating in some of the obviously hopeless cases, particularly those in which the hydrocephalus was associated with a meningomyelocele. In every instance, the parents were acquainted with the experimental nature of the operation and the unpredictability of results, and they either wanted the procedure done to keep their infant alive or to help develop it for others.

Drainage in some manner or another into the peritoneal cavity is an old story and has been reported by Ferguson (1898), Kausch (1905), Cushing (1908), Heile (1910), Hartwell (1910), and also by Davidoff[3] in 1929, who added a thorough historical review of all treatments up to that time.

To the best of our knowledge, Cone[1] of Montreal was the first to use plastic tubes (portex) for the shunt, and one of us (V. R.), who came as a resident from Dr. Cone's service, started this work in our department in January, 1950.

Spitz and Koop,[5] in 1952, presented their observations on 42 cases of hydrocephalus treated by lumbar-peritoneal shunt using a lucite peritoneal button and omentectomy, and reported 73 per cent survivals. They do not operate on infants with noncommunicating hydrocephalus and they have recently modified their technique to include teflon buttons. No data on their recent results are available in the literature as yet.

Data concerning our cases are presented in Tables 1 to 8 inclusive.

TABLE 1

*Type of hydrocephalus**

Noncommunicating	10
Communicating	22
Total	32

* All determined by air injection.

DISCUSSION

Results of Operation and Complications. The infants tolerate the rapid drop in ventricular pressure well, probably because the fluid is reabsorbed through the peritoneal cavity. However, 1 death, from bilateral subdural hematoma, was probably caused by collapse of the brain with rupture of a subdural vein.

* The Torkildsen procedure can be combined with a lumbar subarachnoid to peritoneal cavity shunt to convert a noncommunicating hydrocephalus to a communicating one, and thus avoid placing a subcutaneous tube from ventricle to peritoneal cavity. However, this would require three separate craniospinal operations (skull trephine, suboccipital trephine and hemilaminectomy) as compared with one cranial operation (exploratory trephine) if the tube is inserted into the ventricle and then placed subcutaneously before insertion into the peritoneal cavity. The latter operation would be considerably less shocking to infants. The combined Torkildsen and lumbar procedure could be done as a two-stage operation in infants or for the relief of ventricular obstruction in certain cases of tumor in children and adults.

TABLE 2

Etiology of hydrocephalus

Congenital	
Obstruction at base	13
Obstruction at aqueduct	2
Obstruction at cerebral subarachnoid space	4
Associated with known intracranial hemorrhage at birth	
Subdural hygroma	1
Subarachnoid hemorrhage	1
Intraventricular hemorrhage	1
Postmeningitic	4
Retrolenticular fibroplasia	1
Cervical meningomyelocele	
Obstruction at foramen magnum	1
Cervical meningocele	
Obstruction upper cisterna	1
Lumbar meningomyelocele (Arnold-Chiari)	3

TABLE 3

Age and sex of patients

No. of Patients	Age	No. of Patients	Sex
9	1–3 mos.	16	M
13	4–6 mos.	16	F
8	7–12 mos.		
1	14 mos.		
1	3 yrs.		
Total 32		32	

TABLE 4

*Thickness of cerebral mantle (inner table of skull to ventricle)**

No. of Patients		Thickness of Mantle
10	75%	0.5 to 1.0 cm.
14		1.0 to 2.0 cm.
5		2.0 to 3.5 cm.
3		Undetermined
Total 32		

* From air studies.

TABLE 5

Revisions of operation because of blockage

Type of Shunt	First Operation	No. of Revisions*
Ventriculo-peritoneal	10	13
Subdural-peritoneal	1	2
Lumbar-peritoneal	18	15
Lumbar subarachnoid to lumbar vertebral (hollow screw)	2	4
Ventriculo-sagittal sinus valve	1	1
Total	32	35

* 14 cases, no revisions; 13 cases, 1 to 2 revisions; 5 cases, 3 to 5 revisions.

TABLE 6

Effects from materials used for shunts

Materials*	No. of Times Used	No. of Complications	Longest Period in Peritoneal Cavity Before Revision	Complication
Portex (vinyl plastic)	14	9 (64%)	2½ yrs.	Drying, hardening and breaking of tube from loss of plasticiser (octyl, phthalate), 2 cases. Fibrous capsule intraperitoneal end of tube, 1 case. Tube loose in subcut. tissues, 3 cases. Tube blocked at peritoneal end, 3 cases.
Polyethylene	10	6 (60%)	3 yrs.	Tube blocked by omentum, 1 case. Peritoneal block, 1 case. Broke, 1 case. Kinked, 1 case. Unknown cause, 2 cases.
Stainless steel button (intraperitoneal)	6	3 (50%)	2 yrs.	Tube plugged by omentum, 2 cases; 1 in 2 wks., 1 in 2 mos. Plugged by adhesions, 1 case, 2 wks. after omentectomy (4th op.).
Davol #9 rubber tubing	10	1 (10%)	2 yrs.	Used routinely for intraventricular, subarachnoid or subcutaneous insertions with excellent results. Not used intraperitoneally. Found in contralateral ventricle in 1 case (Fig. 7).
Stainless steel hollow screw. Lumbar subarachnoid space to lumbar bone marrow	2	2 (?)	2½ yrs.	Temporary block revised by portex and latex tube shunt, 1 case 2 mos. and 1 case 1 mo. after 2nd op. Latex tube removed at 3rd op. No definite evidence that screws are working although hydrocephalus under control in 1 case.

* None of the tissues surrounding or blocking any of above materials showed malignant changes.

TABLE 7

Summary of operative procedures and mortality

Total patients operated upon			32
First shunt operation	32		
Total revisions of shunt	35*		
Choroid plexectomy		3	
Lumbar-ureterostomy		4	
Ventr.-ureterostomy		1	
Lumbar-suprahepatic		2	
Revisions in situ		25	
Total Operations			67
Mortality			
Died home after shunt (2 mos.; 6 mos.; 1 yr.; 3 yrs.)		4	
Died home after lumbar-ureterostomy (1 mo.; 6 mos.)		2	
Died hospital after lumbar-ureterostomy		1	
Died hospital after shunt		3	
Died hospital after plexectomy		2	
Case mortality			12 (38%)
Total shunt operations, lumbar or ventr. to peritoneal cavity (ureterostomies and plexectomies excluded)		59	
Died in hospital after shunt		3	
Operative mortality for shunt			3 (5.0%)

*Omentectomies were done in 3 revisions.

TABLE 8

Late results of operation

Total patients operated upon	32	Survived Operation	20 (62%)
Noncommunicating	10	Noncommunicating	4 (40%
Communicating	22	Communicating	16 (73%)

Duration of Followup	No. of Patients	Type of Hydrocephalus Noncom.	Com.	Hydrocephalus Controlled (no. cases)	Mentality of Patients
Less than 1 yr.	4	2	2	3	?
1 to 2 years.	7	none	7	6	Good 2 Poor 5
2 to 3 yrs.	6	1	5	6	Good 2 Poor 4
4 yrs.	3	1	2	2	Good 1 Poor 2

We did not encounter any unusual gastro-intestinal conditions, such as intussusception, serious obstruction of the bowel or diarrhea. Transitory constipation occurred in a few cases.

Abdominal distention developed in 4 cases and it is important to treat this promptly in order to insure continued flow in the system.

In a few cases, there was some temporary edema of the scrotum or mons veneris.

In 3 instances, during revision of shunts, the degree of pressure above which saline would flow into the peritoneal end of the drainage tube was measured with a water manometer. In 2 of these, 50 to 70 mm. of saline was the highest figure obtained with the patient in the horizontal position. During these measurments, respiratory movements were reflected as oscillations in the fluid level of the manometer.

Like other procedures for hydrocephalus, the results are entirely unpredictable, blockage of the system in the peritoneal cavity was frequent and in our group of 32 original operations an additional 35 revisions were necessary (Tables 5 and 7).

The causes for the intraperitoneal blockage were: (1) Plugging by omentum, (2) fibrous capsule or cyst about the tube, and (3) tube cracking or kinking.

Blockage of the tube in the ventricle or subarachnoid space was rarely a problem except in a few instances because of a plug of cerebral tissue or improper insertion.

FIG. 2. Section of portex (vinyl plastic) tubing removed from peritoneal cavity 2½ years after insertion. End of tube in peritoneal cavity was obstructed by a dense, white fibrous capsule which covered it like a stocking. Tubing had lost its plasticiser, was brittle and sharp, and had broken completely at the lumbar end.

Effects from Materials Used for Shunts (Table 6). Portex tubing (vinyl plastic), which we used in the early cases because of its elasticity, lost its plasticiser, hardened, cracked and broke off into the abdominal cavity in 2 cases after 2½ years and in the subcutaneous tissue of the neck in 2 others. Its use, therefore, has been discontinued (Fig. 2).

Polyethylene tubing has been satisfactory in some cases as long as 3 years. It is not as elastic as portex, tends to kink and sometimes crack when bent but does not harden as portex, because it has no plasticiser.

FIG. 3. Hollow stainless steel perforated cylinder (1.25 cm. length×0.1 cm. dia.) inserted through small opening in peritoneum and held by purse-string suture of peritoneum about neck of cylinder below the flange. The flange is sutured to the abdominal muscles, and the elbow (1.5 mm. dia.) is connected to subcutaneous polyethylene or Davol #9 rubber tubing, the other end of which is inserted into lumbar subarachnoid space or ventricle. The cylinder was obstructed by omentum or adhesions in 3 cases. It is working in 3 cases, 2 years after insertion (see Figs. 4–6).

FIG. 4. (*Left*) N.G., aged 2 months, before shunt. (*Right*) Ventriculogram showing marked communicating hydrocephalus (head-hanging view). Lumbar-peritoneal shunt with portex tubing worked well for 2 years and then was obstructed because of breaking of tube.

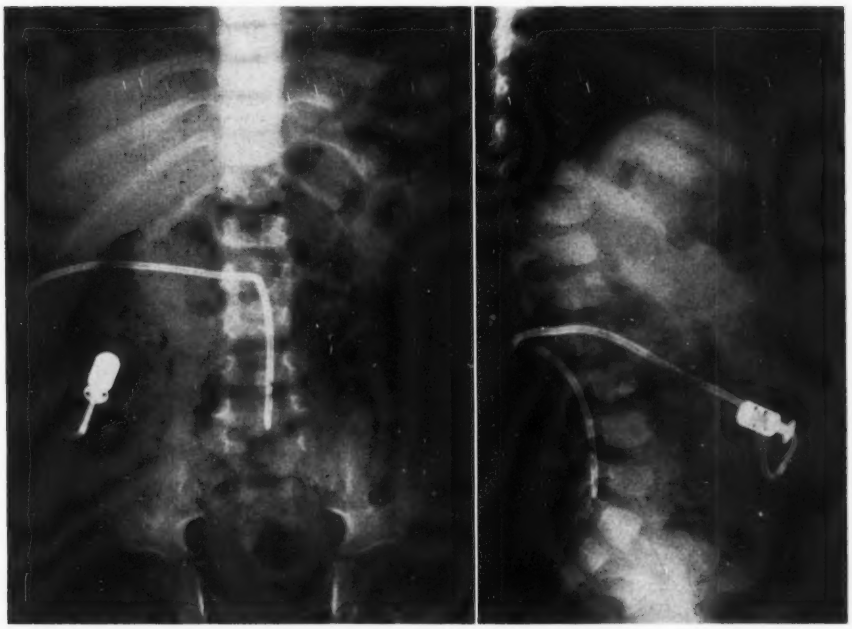

FIG. 5. A-P and lateral roentgenograms of patient in Fig. 4 showing lumbar to peritoneal shunt with rubber tubing and stainless steel cylinder done at 2 years of age and functioning at present.

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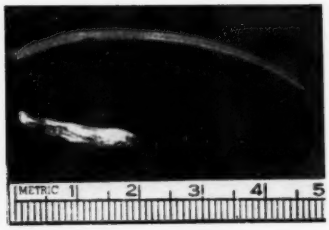

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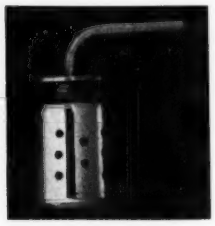

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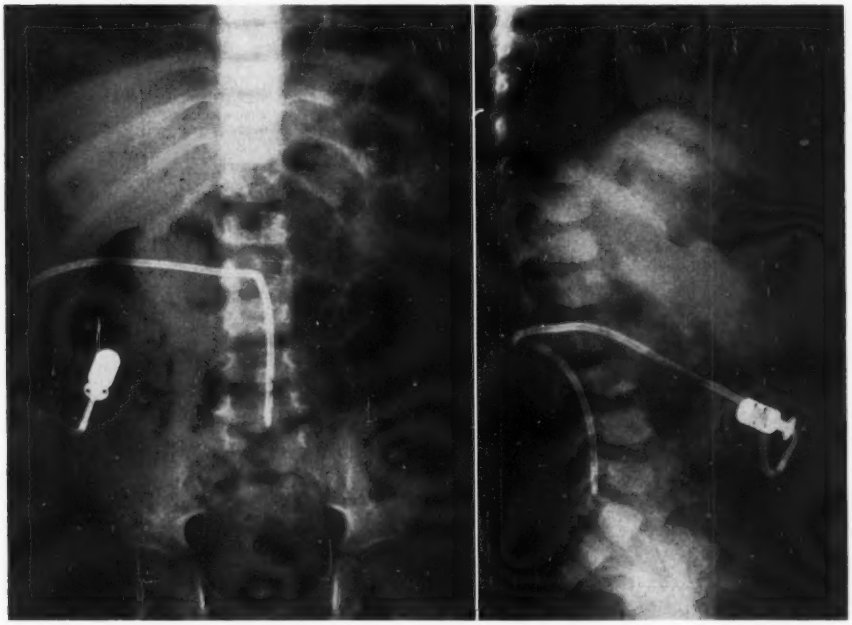

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FIG. 6. N.G., now 4 years of age. He is alert, and appears normal except for large head (see Figs. 4 and 5).

Because of the above problems, we have used a small stainless steel button, suspended through the anterior abdominal wall into the peritoneal cavity like a light bulb in a ceiling (Fig. 3). We connect the extraperitoneal elbow of the button to a piece of #9 Davol rubber tubing which, in turn, is placed subcutaneously in a snake-like pattern (for future growth of patient) and then inserted into the ventricular or lumbar subarachnoid space. Three of these buttons are working satisfactorily 2 years (Figs. 4–6). Two were plugged by omentum within a few weeks after insertion. We believe that omentectomy, as done by Spitz and Koop,[5] may be helpful in preventing this complication in some cases. However, we have done only 3 omentectomies and the followup is inadequate for conclusions.

We now favor #9 Davol rubber tubing for insertion into the ventricles or lumbar subarachnoid space because it has been well tolerated for years in these cavities. We also use it subcutaneously down to the peritoneum because it is flexible, does not kink and can be placed with maximum curvature. We do not use it intraperitoneally but connect it directly to the stainless steel button or

FIG. 7. Unusual complication from subcutaneous rubber tubing: 15 cm. of rubber tubing was inserted into a huge left lateral ventricle through a 2-cm. trephine opening and sutured to dura mater; then it was threaded subcutaneously behind left ear, down lateral neck anterior to clavicle and down thorax to left side of abdomen where it was connected to a 15-cm. length of polyethylene tubing by a small stainless steel tube; the polyethylene tubing was inserted into peritoneal cavity. Symptoms of blockage in 2 months. X-ray of skull shows entire length of rubber and polyethylene tubing in *right* lateral ventricle. The head movements probably acted as a windlass.

anastomose it via a small stainless steel tube to a length of polyethylene tubing, 1 mm. in diameter, which may be inserted into the peritoneal cavity.

In 1 case in which we used the above technique for a left ventriculoperitoneal shunt, blockage occurred in 2 months because of a "migration" of the entire length of tubing into the contralateral ventricle. The material was removed, the choroid plexuses were coagulated and the hydrocephalus was controlled (Fig. 7).

The reaction of the subcutaneous tissues to the Davol tubing is minimal and no different from the capsule surrounding polyethylene. We have never seen any malignant changes in the tissues about it or any of the plastic tubings similar to those reported by Oppenheimer *et al.*[4] when they embedded plastic films and pure polyethylene in rats.

We have tried hollow stainless steel screws inserted through the lumbar subarachnoid space and into the body of the lower lumbar vertebra in 2 cases (lumbar subarachnoid to bone marrow shunt). Although the hydrocephalus is under control in 1 case for a period of 4 years, we are doubtful that the screw is responsible because the hydrocephalus was caused by meningitis and may have resolved spontaneously (Figs. 8 and 9).

In 2 instances, a lumbar subarachnoid to suprahepatic shunt was done

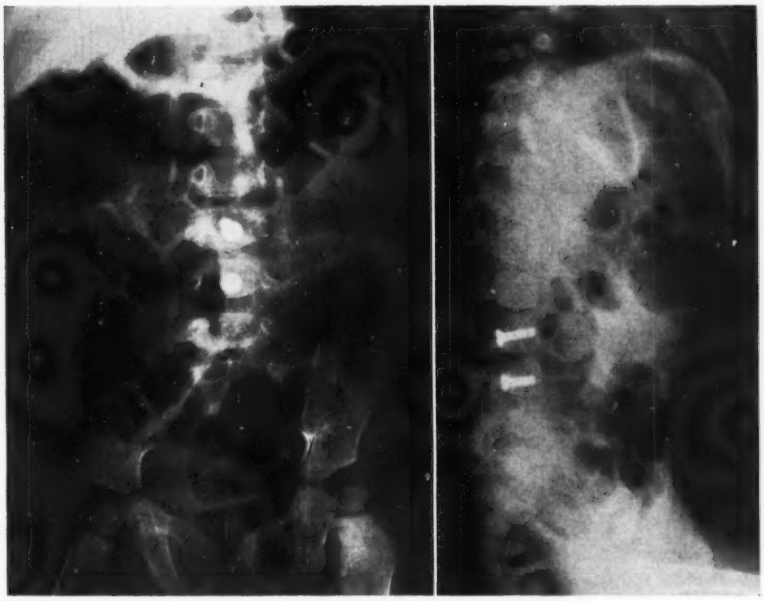

FIG. 8. Hollow stainless steel screws in lumbar vertebral bone marrow used in 2 cases of postmeningitic communicating hydrocephalus. One child is doing well 4 years after insertion but there is no proof that bone marrow absorption is controlling the hydrocephalus rather than spontaneous remission (see Fig. 9).

FIG. 9. X-ray showing marked communicating hydrocephalus before insertion of screws (see Fig 8).

with Davol rubber tubing connected to a polyethylene tube which was placed intraperitoneally over the dome of the liver. In 1 case there was a block by omentum within 3 weeks. The other functioned with intermittent blockage for 1 year and then was blocked completely.

Results of Other Surgical Procedures (Tables 5 and 7). We have done 5 Matson operations: 2 ventriculo-ureterostomies, and 3 lumbar-subarachnoid-ureterostomies.

One patient is doing well 2 months after the procedure. The remaining 4 patients have died: 1 from a malignant liver tumor, 1 month after operation; 1 from a bilateral subdural hematoma which may have been present before the operation, 3 months later; 1 from electrolyte imbalance, 2 months after surgery; and 1 from sudden respiratory paralysis 6 months after operation.

All of the above ureterostomies were done as a "last resort" procedure, following multiple revisions of the peritoneal operations which failed. We do not consider them a fair appraisal of the value of the Matson procedure which should be done as the primary operation for proper evaluation.

CONCLUSIONS

1. A series of 32 infants with progressive hydrocephalus upon whom a shunt operation was done is presented. In 10 cases, the hydrocephalus was noncommunicating and in 22, communicating.

2. Ventriculo-peritoneal and lumbar peritoneal shunts have controlled the hydrocephalus in a *small percentage* (9 per cent of our cases over a maximum followup period of 4 years).

3. The results in communicating hydrocephalus are better than in the noncommunicating type, the survival rate being 73 per cent in communicating and 40 per cent in noncommunicating hydrocephalus during a followup period of less than 1 year to 4 years.

4. The procedure is well tolerated. The operative mortality for 67 operations was 5.0 per cent; the case mortality 38 per cent. The results, however, are entirely unpredictable; blockage of the system is frequent in the peritoneal cavity and in our group of 32 original operations, 35 re-operations were necessary for this complication.

5. Portex tubing or any plastic tubing containing a plasticiser should be avoided because of a tendency of these substances to later become brittle and crack.

6. Polyethylene is preferable but has the disadvantage of kinking when bent.

7. A stainless steel peritoneal button has been satisfactory in 50 per cent of the 6 cases in which it was used. However, it was obstructed by omentum in 2 cases. Omentectomy, as advocated by Spitz and Koop, might increase the efficiency of this button.

8. Davol #9 rubber tubing is extremely well tolerated in the ventricles and spinal subarachnoid space and shows only a slight capsular reaction in the subcutaneous tissues. At present, we believe that it is the material of choice for the extraperitoneal portion of the shunt.

9. The operation of ventricular or lumbar peritoneal shunt should not be abandoned. As stated previously, the procedure is a simple one; the operative mortality is low and some lives are prolonged.

In some postmeningitic cases, the procedure may prevent severe brain destruction during the acute stage of the block and carry the patient until adequate pathways of fluid absorption are established and spontaneous remission occurs.

We believe there may be other factors contributing to blockage in addition to the usual peritoneal-omental reaction to foreign material. The constant drainage of ventricular or subarachnoid fluid might well result in prolonged negative pressure in the ventricular or subarachnoid part of the shunt and positive pressure in the intraperitoneal portion of the shunt which could force omentum or bowel against the tube or button for a period long enough to permit adhesions to take hold.

Some mechanism (perhaps a valve) that would keep the ventricular or subarachnoid pressure just above that of the peritoneal cavity might reduce the incidence of blockage in these shunts. We are investigating this factor.

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CASE REPORTS AND TECHNICAL NOTE

SPINAL EXTRADURAL CYSTS

REPORT OF A CASE

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Compression of the spinal cord by an extradural cyst is a relatively rare lesion. Elsberg, Dyke and Brewer[3] were the first to describe such a lesion, reporting 4 cases. The original description can hardly be improved upon:

"The individual is an adolescent with the history and symptoms of a progressive spastic paraplegia. Pain is absent or is not a prominent symptom. The objective disturbances of sensibility are slight and their upper level is in the mid-thoracic region, usually at the sixth or seventh thoracic dermatome. The manometric tests demonstrate a subarachnoid block with the characteristic spinal fluid changes of cord compression. Measurements on antero-posterior x-ray films show that the interpediclar spaces of three or more vertebrae somewhere between the fourth and the tenth thoracic vertebrae are enlarged. The pedicles of the affected vertebrae, especially those of the sixth, seventh and eighth, are narrowed and atrophic."

Lehman[9] reported 2 cases; Mixter[12] another. Cloward and Bucy[2] described 2 cases, one of which was associated with kyphosis dorsalis juvenilis, and made a critical review of all former cases that had been studied. Peet and Kahn,[13] Swanson and Fincher,[14] and Haffner[5] subsequently presented additional cases. Jacobs, Smith and Van Horn[7] were the first to report a case in which a communicating cyst filled spontaneously from the subarachnoid space during myelography. The author's case, likewise, demonstrated such a communication. Hyndman and Gerber,[6] Adelstein,[1] Turnbull,[15] Krauss,[8] Good, Adson and Abbott,[4] and Mayfield and Grantham[10] have all reported additional cases.

CASE REPORT

C.E.W., a 48-year-old welder, was first examined on Nov. 19, 1953 complaining of pain in the left low back and hip, which radiated down the left leg to the foot. On June 12, 1951 he had stepped off a truck and slipped on a small rock and had fallen, spraining his right ankle. Because of pain, he was disabled, except for light work.

Two weeks after his injury, intermittent pain appeared radiating into the left hip and down the left leg. In May, 1953 he became aware of some numbness of the left leg and foot. By July, 1953 some weakness of the left leg with accompanying atrophy of the muscles had appeared. Coughing, sneezing or straining did not aggravate the pain. However, standing for long periods of time caused his low back and left leg to ache. He favored his left leg when walking and could not walk more than one-half mile. Following exercise his left leg became so tired he could hardly move it.

Examination. The right calf measured 37.3 cm., the left 34.0 cm. in circumference. All the muscles of the left lower extremity were weaker than those on the right. This weakness applied

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to inversion, eversion, plantar flexion, and especially dorsiflexion of the left foot. His gait was spastic with the left leg. He bent forward until his fingertips came within ten inches of the floor. Backward bending, as well as bending to the right and left, were only slightly restricted. All back bending motions caused only slight discomfort in the region of the left hip.

The left gluteus maximus muscle was more flaccid than the right. No tenderness could be made out by heavy pressure or percussion anywhere along the entire spine. A slight degree of hypesthesia existed to temperature and pain below the 8th, or possibly the 9th, dorsal cord segment; this seemed more marked on the left than on the right. Vibratory sensibility was decreased in the left leg below the knee. Light touch and position sense were everywhere preserved.

Patrick's test caused some discomfort in the left hip region. Increasing the intra-abdominal pressure produced pain in the left hip. Straight leg raising could be carried to 80°, right and left. Lasègue's sign was bilaterally negative.

Superficial abdominal and epigastric reflexes were absent on the right, sluggish on the left. Deep reflexes of both upper extremities were moderately active and equal. Patellar and knee jerks were much quicker on the left than on the right, almost to the point of spasticity. Deep tendon reflexes were active and equal, the left perhaps a little quicker than the right. Chaddock and Babinski signs were strongly positive on the left, negative on the right. There was no ankle clonus.

Electromyography on Nov. 19, 1953 revealed no fibrillation of denervation. All motor unit activity was normal.

Radiological Studies. Roentgenograms made previously on Nov. 4, 1952 revealed a moderate scoliosis centering at the 2nd lumbar interspace with the convexity to the right and with marked narrowing of the 4th and 5th lumbar interspaces. There was considerable eburnation in the bone adjacent to the articular facets on the right between the 3rd and 4th and the 4th and 5th lumbar vertebrae.

Pantopaque myelography on Nov. 5, 1952 showed a shallow indentation on the left side at the 4th lumbar interspace, which was considered to be related to the scoliosis of the lumbar spine and which centered at that point. It did not seem to represent a protruded intervertebral disc. Spinal fluid examination at that time revealed a clear, colorless fluid, with 3 cells and total protein of 50 mg. per cent.

Two points are worth mentioning regarding these previous studies. First, the x-rays did not include the dorsal spine, which would have revealed the pathology; and, second, the pantopaque was not allowed to pass upward into the mid-dorsal region.

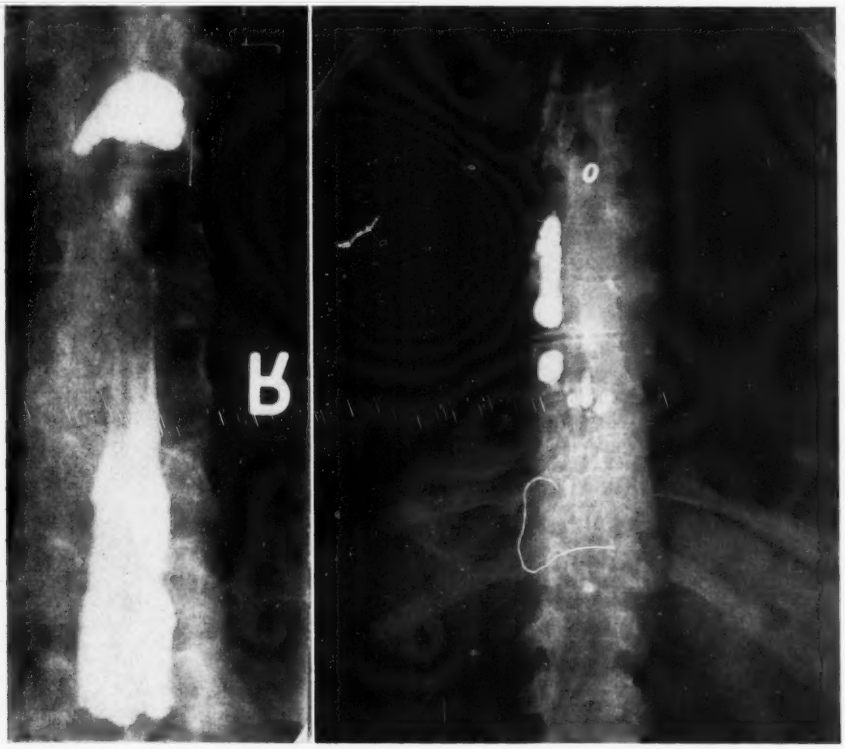
A lesion of the spinal cord was suspected. Former examiners leaned toward a diagnosis of degenerative disease of the central nervous system, such as multiple sclerosis.

On Feb. 3, 1954 views of the dorsal spine were performed. These showed the pedicles in the 6th, 7th and 8th dorsal vertebrae to be absent (Fig. 1). The 7th dorsal vertebra

FIG. 1. Roentgenogram showing widening of the spinal canal with absence of the pedicles of the 6th, 7th and 8th dorsal vertebrae.

showed evidence of erosion in the posterior aspect of the vertebral body. There was a moderate scoliosis in the dorsolumbar spine centering at the 1st lumbar interspace with the convexity to the right.

A pantopaque myelogram on Feb. 3, 1954 showed a complete block at the 5th dorsal interspace (Fig. 2). Below the level of the block was a large filling defect, extending down to the level of the 8th dorsal interspace. This filling defect caused the pantopaque to flow around its right lateral aspect (Fig. 3). After the patient was placed in the erect position, most of the



FIGS. 2 and 3. (*Left*) Myelogram showing complete block of pantopaque at the 5th dorsal interspace with large filling defect below, extending to the level of the 8th dorsal interspace. (*Right*) Pantopaque remaining at the 8th dorsal interspace lateral to the midline with patient in the upright position.

pantopaque flowed back to the lumbar area, but a small quantity remained at the level of the 7th dorsal interspace. These findings suggested a large space-occupying lesion in the spinal canal extending from the level of the 5th through the 8th dorsal vertebrae. In view of the destruction of the pedicles in the 8th dorsal vertebra, the lesion undoubtedly extended down to this point and was consistent with a large tumor or possibly a cyst.

The spinal fluid was clear and colorless, containing 4 cells and total protein of 40 mg. per cent.

Operation. On Feb. 17, 1954 laminectomy revealed an extremely large extradural cyst, which was encapsulated by a fairly thick membrane. The cyst extended from the level of the 5th through the 8th dorsal vertebrae. The spinal canal in this area was enlarged to almost

three times its normal diameter. The cyst was retracted medially and beneath it lay normal dura mater covering the spinal cord. A small neck of the cyst was present at its distal aspect, which communicated with the subarachnoid space. The cyst was completely removed at the neck, which was ligated. The cyst measured 9 cm. in length and 3×2 cm. in its greatest transverse and anteroposterior diameters.

Microscopic Examination. The cyst wall was composed of laminated acidophilic, poorly cellular connective tissue. In places there was mild infiltration by mononuclear cells. The lining of the cyst was devoid of epithelium or other characteristic cells. Within the wall of the cyst there were aggregates of pigment-bearing cells. The blood vessels were rather dilated and somewhat congested. A few hemorrhagic areas were seen. Diagnosis: "Cyst wall containing pigment-bearing cells showing infiltration with lymphocytes. Fatty tissue."

Course. Following surgery, although evidence of spasticity when walking still existed, the left Babinski sign remained strongly positive.

DISCUSSION

Symptomatology and Diagnosis. Spinal extradural cysts rarely cause pain and, when present, the pain is usually slight. Weakness of the lower extremities, associated with difficulty in walking, may often progress to a spastic paraplegia. Loss of sensation may or may not be present, and is never severe. Disturbance of bowel and bladder function may appear early or late. Remission of symptoms may occur and clinically the course is suggestive of, and may be confused with, multiple sclerosis or similar degenerative diseases of the central nervous system. Vasomotor changes are uncommon. Light touch, temperature and pain sensations are usually only slightly decreased below the level of the lesion. Position and vibratory sensibility may be severely involved or totally absent in the lower extremities.

Symptoms have appeared in patients from $8\frac{1}{2}$ years to 46 years of age. The duration of the neurologic symptoms varied from 2 months to 13 years. A progressive paraplegia was the rule. One patient showed evidence of the disc syndrome with radicular pain.

Roentgenological Findings. Widening of the spinal canal in either diameter and/or erosion of the pedicles at the level of the cyst are usually present. The vertebrae in the involved area show an epiphysitis with irregularity of the epiphyseal plates. Kyphosis may or may not be present. Most of these cysts occur in the thoracic region, only a few in the lumbar region. None has been reported in the cervical region, with the exception of a traumatic cyst described by Meredith[11] in 1940. He reported 9 cases of unusual tumors and tumor-like lesions of the spinal canal. His first case was that of an extradural cyst at the 7th cervical vertebral level. This was described as an extension of herniated arachnoid through a small puncture opening in the dura mater. The traumatic origin was suspected in view of the history of a severe contusion of the cervical spine several months before onset of symptoms. This was substantiated by the pathologist's finding of hemosiderin in the extradural tissue removed, and a fragment of old necrotic bone. These cysts rarely communicate with the spinal subarachnoid space. There may, or may not, be a complete block to pantopaque myelography at the level of the lesion.

Etiology. Most writers consider these cysts to represent a diverticulum of the spinal meninges, the cysts being primarily filled with cerebrospinal fluid. Cloward and Bucy[2] suggested that the pedicle of the cyst may become occluded and the cyst continue to grow by virtue of the intrinsic secretion from its walls. The case reported by Jacobs, Smith and Van Horn[7] and that of the author demonstrate that these cysts may communicate with the spinal subarachnoid space. Others, at surgery, have

reported communication of the cyst with the spinal subarachnoid space. This was demonstrated by compression of the jugular veins, which caused the cyst to fill or empty, depending on the degree of compression of the jugular veins. The cyst is considered to be an evagination of both the dura mater and the arachnoid which, because of the intracystic pressure, have fused into one membrane. Why these cysts almost always appear in the lower mid-thoracic region is not understood. The constant pressure exerted by the cyst no doubt disturbs the circulation of the involved vertebral bodies more in some patients than in others and may be responsible for the kyphosis that sometimes occurs.

SUMMARY AND CONCLUSION

A case of spinal extradural cyst is reported. The diagnosis may be suspected roentgenographically if widening of the spinal canal and erosion of the pedicles are present. This case emphasizes the value of a thorough radiographic survey of the vertebral column in cases which seem to present atypical findings of degenerative diseases of the central nervous system. The early recognition of such lesions, followed by prompt surgery, may prevent permanent spinal cord damage. Young children and adolescents who present kyphosis that cannot be satisfactorily explained should be suspected of harboring such a lesion.

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THROMBOSIS OF THE INTERNAL CAROTID ARTERY CAUSED BY A CERVICAL RIB

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The neurological symptoms caused by thrombosis of the internal carotid artery have been fully discussed in many articles during the last few years.[1-3] However, in the literature at my disposal I do not find any reference to thrombosis of the internal carotid artery caused by the presence of a cervical rib on the same side. So I venture to report the following case which came under my care recently.

CASE REPORT

A.S., a female aged 23 years, began to have intermittent attacks of pain in the right arm together with numbness of all the fingertips of the right hand. Subsequently the arm became cold and clammy, and wasting became gradually apparent. Five months after the onset of symptoms the patient was suddenly taken ill. She lost consciousness and displayed convulsions on the left side. The seizure lasted almost an hour and the patient was left with a left-sided hemiplegia, which gradually improved during the following months.

Examination 3 months after the attack disclosed a woman in good general health. The pulse rate was 76/min. on the left but could not be felt on the right side. B.P. was 130/70. The optic fundi and visual fields were normal. In the right supraclavicular groove an abnormal rib could be felt as a bony prominence.

The muscles of the right arm, especially the thenar and hypothenar, were atrophied and all tendon reflexes were absent. The right hand was cold and the radial pulse could not be felt. Sensations to pinprick and temperature variations were diminished up to region of the elbow. On the left side a spastic hemiparesis was present with partial contracture of the fingers. Tendon reflexes were all exaggerated. Hofmann's reflex was positive, but plantar reflexes were flexor.

FIG. 1

Roentgenograms verified the diagnosis of a cervical rib (Fig. 1).

Carotid angiography was performed on the right side by the open method; 12 cc. of 35 per cent Nosydrast were injected into the common carotid artery while the needle was kept in the direction of the internal carotid artery. On the lateral view (Fig. 2) all the branches and ramifications of the external carotid were filled but the internal carotid became gradually thinner and disappeared at a distance of about 4 cm. above the bifurcation.

Operation. Under general anaesthesia the cervical rib was exposed through an anterior incision. The subclavian artery was found to be thrombosed. The rib was removed in pieces and the brachial plexus was relieved from pressure. The stellate ganglion was surrounded by adhesions to such a degree that it could not be clearly identified.

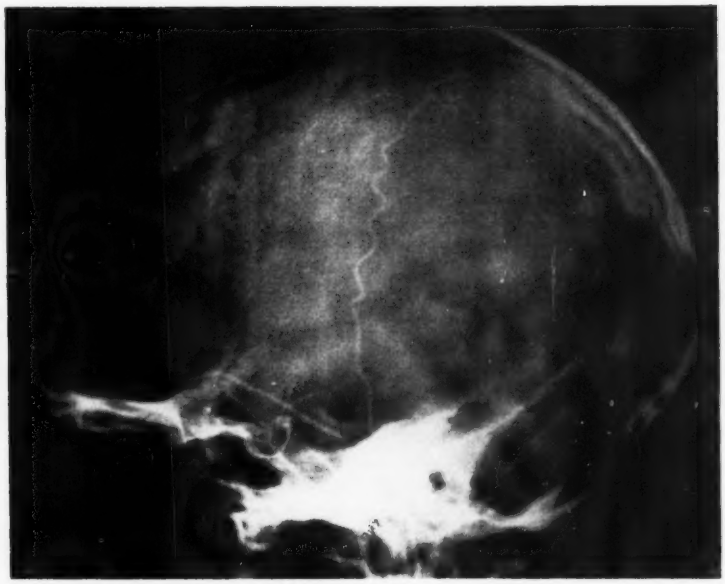

FIG. 2

Postoperative course was uneventful. About 16 days after the operation the right hand started to feel warmer and gradually returned to normal colour.

COMMENT

Various etiological factors in nontraumatic thrombosis of the internal carotid artery have been discussed: arteriosclerosis,[2,3] thromboangiitis obliterans,[4] acute infections,[3] syphilis,[4] and pathological conditions of neighbouring tissue.[4] Less attention has been paid to the part that the sympathetic nervous system plays in the etiology of this disease. Only Fisher[2] referred to this question. He wrote: "The rôle of sympathetic impulses, normal or abnormal, in producing vasoconstriction can only be surmised, but it is not unlikely that they play an important part." It is well known that a cervical rib sometimes leads to extensive adhesions in the so-called cervico-axillary groove. In the case reported here it is believed that continued irritation of the sympathetic ganglions, caused by adhesions, had led to chronic spasm of the carotid artery with its subsequent thrombosis.

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TWO CEREBELLAR TUMOURS WITH UNUSUAL FEATURES

1. CYSTIC ASTROCYTOMA. 2. PAPILLOMA OF THE CHOROID PLEXUS

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From time to time cases occur that are so outside usual experience as to be worthy of record. In the two cases presented here, one tumour was a cerebellar astrocytoma of long symptomatology and the other an intracerebellar papilloma of the choroid plexus.

CEREBELLAR ASTROCYTOMA

Case 1. A 29-year-old male was admitted to Dunedin Hospital on July, 10 1952, complaining of left frontal headaches for 3 months and vomiting associated with sweating and a feeling of fever. There had been weakness and tinglings of both hands for 3 weeks and double vision for 1 week.

His mother told us that at the age of 7 years he had fallen off a gate and had been unconscious for a few minutes. Two months later his sight became poor, he had double vision and a squint was noticed. He was very unsteady on his legs. A tonsillectomy was done. Within a month of this headaches developed and his legs were worse. He was admitted to hospital and was thought to have poliomyelitis. He was soon sent home and from then until the age of 17 years he was a semi-invalid. He spent a lot of time in bed because of unstable limbs and difficulty in controlling his bladder. At the age of 17 years he began to improve. Although unsteady on his legs he was able to earn a living as a dustman. He had kept at work until the onset of his present symptoms.

Examination. He was retarded mentally, apathetic and rather drowsy. The vision was J.16 in either eye. There was bilateral papilledema with secondary optic atrophy. He had bilateral ptosis which was greater on the right side. The left external rectus muscle was weak. There was a right facial weakness and the tongue deviated to the right. All his limbs were severely ataxic. Only the ankle jerks were present and both plantar responses were extensor. The neck was stiff.

Lumbar puncture gave a pressure of over 300 mm. water. The fluid contained 165 mg. protein per cent. Wassermann reaction was negative and Casoni test was positive. Radiographs of the skull showed erosion of the dorsum sellae.

Operation. On July 14, 1952 a ventriculogram was done. This showed hydrocephalus with a dilated aqueduct displaced forwards.

The same day the posterior fossa was explored. There was a brown mass between the cerebellar tonsils. The vermis above contained a cyst from which 40 cc. of yellow fluid were aspirated. The mural tumour of the cyst was removed completely.

Course. He was able to leave hospital on Sept. 4, 1952. There was severe optic atrophy and the vision was J.16 on the right and J.20 on the left. He had a slight nystagmus to the right and a right facial weakness. He could walk with aid of a stick but had considerable ataxia of the arms and legs. The plantar responses were extensor.

At follow-up on Sept. 16, 1953 he was not at work but was able to dig the garden. There was only slight ataxia of the arms but his gait was moderately unsteady.

Pathological Examination (Dr. R. Rodda). The tumour mass measured 4×3×3 cm. and weighed 15 gm. Histologically the neoplasm was highly vascular with extensive areas of necrosis. In the more cellular areas it comprised well differentiated astrocytes—many plump and some fibrillary in type. The tumour was an astrocytoma (Kernohan grade 2).

INTRACEREBELLAR PAPILLOMA OF THE CHOROID PLEXUS

Case 2. A 67-year-old male was admitted to Dunedin Hospital on Sept. 1, 1952, in a severe confusional state. His relatives said that on July 10, 1952 he had had a short period of unconsciousness but had been able to drive his car home afterwards. His speech had been slurred and some drooping of the left face had been seen. A month later headaches, vomiting and slurred speech developed. He became confused and incontinent. On Aug. 22, 1952 he was admitted to a private hospital in Wellington under the care of Dr. C. Burns, where it was found that he was facile and had a poor memory. Papilledema was present. There was nystagmus in all directions. The speech was slurred. He could not stand alone. The plantar responses were flexor.

Examination. He was grossly confused. The speech was slurred and partly jargon. He was unco-operative and incontinent. There was bilateral papilledema. The left arm and leg were ataxic and weak. The tendon reflexes were equal and the plantar reflexes were flexor.

Radiographs of the skull and chest were normal. A right carotid arteriogram showed a hydrocephalus.

Operation. On Sept. 9, 1952 a ventriculogram was done. There was a hydrocephalus and the fourth ventricle was displaced to the left.

This was followed by a posterior fossa craniotomy. The right cerebellar hemisphere was enlarged but there was no other external evidence of a tumour. A cyst was tapped at a depth of 2.5 cm. and this contained 30 cc. of yellow fluid with a protein content of 380 mg. per cent. The cyst was opened. The wall was composed of soft reddish material and this was removed. A smear biopsy (Dr. R. Rodda) was suggestive of a secondary carcinoma.

Course. After the operation there was some temporary improvement but he slowly deteriorated and died on Sept. 13, 1952.

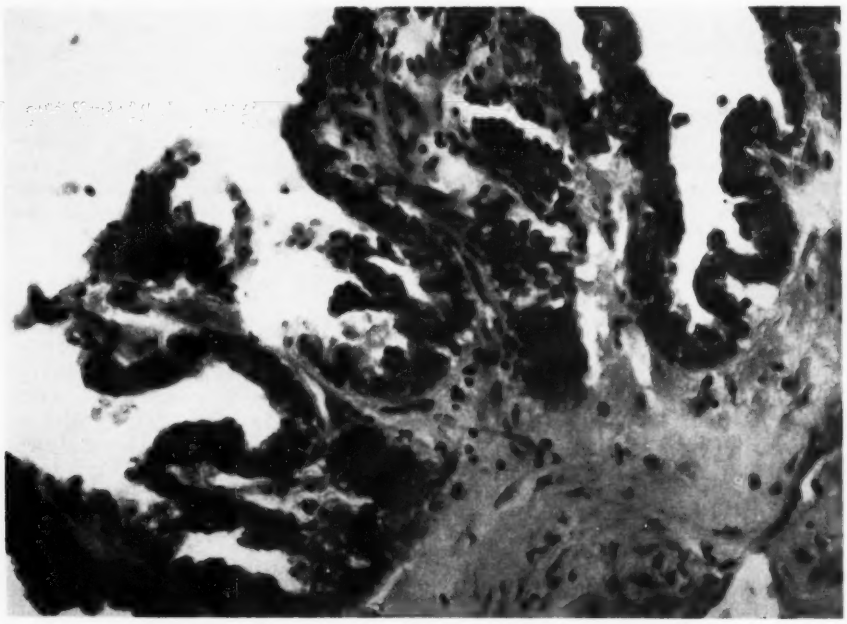

FIG. 1. Photomicrograph of papilloma of choroid plexus removed from the cerebellum (haematoxylin and eosin, ×140).

Pathological Examination (Dr. R. Rodda). The material removed at operation showed papillary processes covered by cells resembling choroid plexus. There was no evidence of malignancy. The tumour was a choroid plexus papilloma (Fig. 1).

A full *autopsy* was done and after extensive examination, the abnormalities were found to be solely in the brain. The brain weighed 1580 gm. There was tonsillar coning. There was a surgical cavity in the right cerebellar hemisphere, that measured 3 cm. across. The softening around the site of operation involved the dentate nucleus. There was no macroscopic connection with the fourth ventricle (Fig. 2). Coronal slices of the cerebral hemispheres showed a marked hydrocephalus. Blocks of tissue from the depths of the operative field showed no histological evidence of any choroid plexus papilloma or any connection with the fourth ventricle or its contents.

FIG. 2. Section through cerebellum and pons showing the site of the tumour.

DISCUSSION

A duration of 21 years from the first symptom to removal is remarkable for cerebellar astrocytoma. The usual length of symptoms for a cystic midline cerebellar tumour was given as 31 months by Elvidge *et al.*,[4] and in 1 case the duration was 10 years. Mabon *et al.*,[7] gave a pre-operative course of 13.9 months for grade 1 astrocytoma and of 11.7 months for grade 2 tumours. The average length of symptoms in Tola's[9] series was 16.5 months with a maximum of 86 months.

There are several cases of prolonged symptomatology. One of Cushing's patients (Case 5[3]) with a right cystic cerebellar tumour had symptoms for 13 years before aspiration was done through a burr hole. The cyst was re-aspirated 11 years later and another 2 years elapsed before the tumour was removed. Bucy and Gustafson[1] had 1 case in which a cyst was aspirated and there was no recurrence over the next 8 years. Hausman and Stevenson[6] described a case in which the total duration of symptoms was 45 years. No operation was performed and at autopsy a cystic astrocytoma of the vermis was found.

It seems that some arrest in the evolution of cystic astrocytomas of the cerebel-

lum can occur and they may remain quiescent for years. Mabon *et al.*[7] found that in their grade 1 astrocytomas the presence of a cyst added only some 8 months to the average survival time of 78 months after operation. It cannot be entirely without significance that these tumours of long prodromata were all cystic. Cushing[3] had many cases of long survival following the removal of the mural tumour.

Papillomas of the choroid plexus constitute 0.4 per cent of verified brain tumours in Olivecrona's[2] series of 4185 cases. Of these 17 papillomas, 10 were in the posterior fossa and made up 1.0 per cent of his posterior fossa tumours.

Sometimes the tumour presents as an intracerebellar mass but connection with the choroid plexus can be demonstrated—usually at autopsy. Greene[5] published a case in which no connection between the choroid plexus and the intracerebellar papilloma could be found. This was the first such instance, as far as is known, and the present case is the second. In both instances the tumours were cystic and occurred in the elderly. Papillomas of the choroid plexus are rare in patients over the age of 60 years.[8] Such a diagnosis cannot be accepted at this age unless an autopsy excludes the presence of a primary neoplasm elsewhere.

SUMMARY

1. A case of cystic astrocytoma of the cerebellum with 21 years' prodromata is described.

2. A case of an intracerebellar papilloma of the choroid plexus without connection with the choroid plexus is described.

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POSTERIOR INFERIOR CEREBELLAR ARTERY SYNDROME

FOLLOWING A FRACTURE OF THE CERVICAL VERTEBRA

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The syndrome of the posterior inferior cerebellar artery was described by Wallenberg[8] in 1895, and subsequently recorded by many other authors.[4,5,7,9] The symptom complex is usually typical and is characterized by a sudden onset of decreased pain and temperature sensitivity on the side of the body contralateral to the lesion and disturbance of these same modalities of sensation on the side of the face ipsilateral to the lesion. Generally there is no motor involvement except a slight transient weakness of the extremities on the ipsilateral side. There may be an ipsilateral Horner's syndrome, ipsilateral involvement of the 5th through the 12th cranial nerves, ataxia, and nystagmus. Loss of consciousness is unusual.

The usual cause of occlusion of a posterior inferior cerebellar artery is considered to be arteriosclerosis. Goldstein[2] described 6 cases with this as the etiologic factor and there are many more similar reports in the literature. Davison and Spiegel[1] presented 2 cases in which the occlusion was attributed to metastatic carcinoma, primary in the lung.

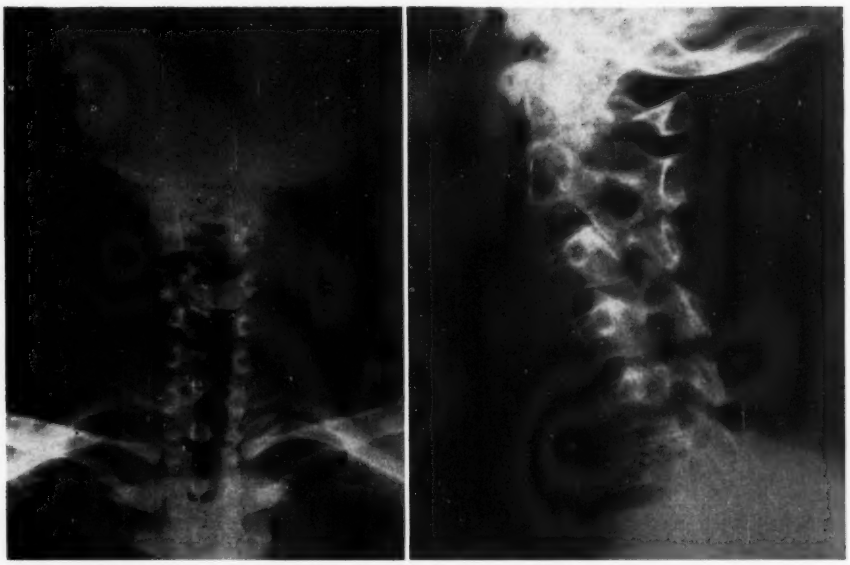
The purpose of this report is to present a case in which the syndrome occurred as a result of skeletal traction for a fractured cervical vertebra. No case with similar etiology was found in a review of the literature.

CASE REPORT

A 29-year-old robust police officer was admitted ambulant to the hospital on July 20, 1954, 18 hours after a diving accident. His only symptom was numbness of his right thumb. Physical and neurological examinations at this time revealed no abnormality except for spasm of the muscles of the neck. Roentgenograms of the cervical spine showed a fracture of the lateral mass of the 5th cervical vertebra on the right (Fig. 1). The 5th cervical was dislocated on the 6th cervical vertebra (Fig. 2).

Crutchfield tongs were applied and 5 pounds of traction was used. The patient did well for 4 days when he suddenly cried aloud because of bilateral facial pain. He also complained of a transient numbness of the right side of his body. Traction was removed and his complaints were immediately relieved. A few hours later skeletal traction was reapplied and the patient remained asymptomatic until the 8th of August. At this time he complained of a vague discomfort over the entire left side of his body accompanied by nausea and vomiting. At no time did he lose consciousness. Neurological examination at this time revealed a right Horner's syndrome, an absent right corneal reflex, an hypoesthesia over the right side of his face in the distribution of the 2nd and 3rd divisions of the trigeminal nerve and of the left side of his body from the 3rd cervical dermatome caudad, a rotary nystagmus, a right 6th nerve palsy, slurred speech, difficulty in swallowing, and a weakness of the right hand. The Crutchfield tongs were removed.

Three days later the neurological signs and symptoms began resolving. He could now talk and swallow without difficulty. The diplopia and the weakness of the right hand disappeared. Four weeks after this episode examination revealed a mild hypoesthesia on the left side of his



FIGS. 1. and 2. Roentgenograms showing (*left*) fracture of lateral mass of the 5th cervical vertebra on the right and (*right*) forward displacement of the 5th on the 6th cervical vertebra.

body and on the right side of his face. The Horner's syndrome and the involvement of the other cranial nerves had disappeared. He was given a cervical fixation brace and was discharged from the hospital.

DISCUSSION

The symptoms presented by this patient correlate well with those of the syndrome of the posterior inferior cerebellar artery. It is believed that the traction caused compression of the vertebral artery with resultant ischemia of the area supplied by the posterior inferior cerebellar artery. Goodhart and Davison[3] discussed the vascular anatomy in this region and concluded there are many variations of the posterior inferior cerebellar and vertebral arteries. Ramsbottom and Stopford[6] stated that the syndrome is easily recognized and the anatomical location of the lesion is usually well defined but that it is very difficult, if not impossible, to state with certainty which of these two vessels is involved. This patient's cervical fracture was on the same side as his thrombosis and in all probability the traction applied to his neck in some way decreased the circulation in the vertebral artery or stretched the posterior inferior cerebellar artery, thereby producing an occlusion. The use of traction must have been implicated in some manner since its removal reversed the acute symptoms on two occasions. Direct compression or trauma from the fracture to the right vertebral artery can be dismissed since one would expect symptoms almost immediately which this patient did not have. There was no evidence of any of the usual common etiological factors such as arteriosclerosis or metastases.

SUMMARY

A case is reported of a fracture dislocation of the 5th on the 6th cervical vertebra in a patient in whom a posterior inferior cerebellar artery syndrome developed following skeletal head traction. No similar etiological factor for this syndrome was found in a review of the literature. The mechanics presented by this case are discussed.

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HETEROTOPIC BRAIN TISSUE AS AN ISOLATED EMBRYONIC REST

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A single case history is worthy of report if it presents a previously unrecognized condition or if it sheds further light on unproven concepts of pathogenesis or treatment. The present case seems to fulfill both criteria.

Congenital cutaneous defects of the scalp are quite rare. When they occur they appear as variously discolored areas completely devoid of hair, usually over the vertex of the head. They range in size from circular lesions 1–2 cm. in diameter to large defects embracing both anterior and posterior fontanelles and a narrower area of the scalp between. Usually there is no associated defect in the underlying skull, but in a number of instances such a defect has been present and, in some, there may be a direct connection with a major blood vessel such as the sagittal sinus.

Though less common than those in the scalp, congenital cutaneous defects have been observed in many parts of the body including the trunk and extremities, and such lesions are usually neither bilaterally symmetrical nor localized to the midline. The etiology is unknown and may not be the same in all cases, but the previously accepted theory that they result from inflammatory adhesions between the amnion and body surface has been largely discarded. Whatever their origin they probably differ from the midline defects which occur anywhere from the cranium to the sacrum.

An associated lesion which has recently received wide recognition in British and American literature is the congenital dermal sinus which connects the body surface with the meninges or central nervous system. It is possible that many of the reported cases of isolated cutaneous midline defects may have had such a connection which was overlooked. Dermal sinuses are presumably related to the case under consideration.

In the present instance the lesion was believed, pre-operatively, and even at operation, to be an isolated congenital cutaneous defect without any transcranial connection. The correct diagnosis, derived from microscopic examination of the tissue, was unexpected. A reasonably comprehensive search of the literature revealed no identical case, but there can be no doubt that there is a direct correlation between this and certain previously recognized lesions. The case is therefore of interest with respect to differential diagnosis and management, and it is pertinent to some currently unsolved problems in the fields of embryology and neuropathology.

CASE REPORT

Children's Hospital #88886. C.W., a 1-year-old white female infant, one of dizygotic twin children (male and female) of a physician, was born March 3, 1952. At birth she was observed to have a flat circular bluish-red lesion in the posterior midline of the scalp, approximately over the lambda. Neurosurgical consultation and detailed roentgenological study of the skull, shortly after birth, led to the conclusion that the lesion was not a meningocele or encephalocele and that there was no collateral defect in the skull or cerebral cortex. No immediate

treatment was advised and the infant's subsequent growth and development were normal in every way, paralleling those of her twin brother.

She was first seen on Feb. 24, 1953, when she was nearly a year old. Her physician-father reported that there had been no evidence of independent growth and that the lesion had increased in size only in proportion to the growth of the child.

Examination. The general appearance of the lesion was as shown in Fig. 1A. It was symmetrical and almost perfectly circular, about 3.25 cm. in diameter, and lay slightly anterior to the lambda. The color was dull red, with a slight pallor at the extreme periphery, and the entire lesion became somewhat cyanotic when the patient cried. It was not spongy or compressible, however, and though it blanched slightly under pressure, the color returned promptly and there was no clinical evidence of excessive vascularity. The growth of hair in the surrounding scalp was normal, and terminated abruptly at the periphery of the lesion, the surface of which was generally flat, with a small nodular elevation 0.5 cm. in diameter near the center. It was covered with a dry epithelium-like surface, without ulceration, tenderness, or evidence of existing or previous inflammation.

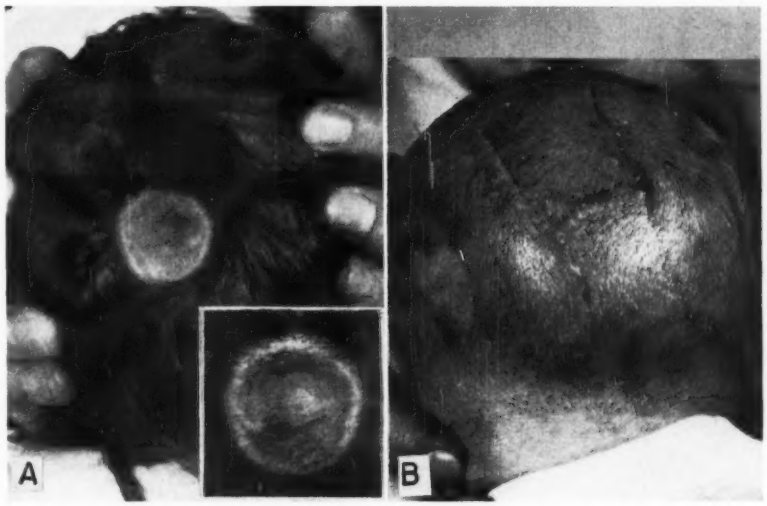

FIG. 1. (A) Gross appearance of the lesion prior to operation (see text for detailed description). (B) Appearance of wound on 11th postoperative day, when patient was discharged from hospital.

Aside from this single isolated lesion in the scalp, the physical examination and routine laboratory studies revealed no abnormalities and the child appeared to be perfectly healthy.

Operation was performed on March 20, 1953. The initial step in the procedure consisted of simple excision around the margin of the lesion, leaving a circular defect slightly over 4.0 cm. in diameter. The underlying skull was well exposed, and no demonstrable defect was recognized. Using parallel tangents to the circular wound, double sliding flaps were constructed, slightly wider at their bases, to provide maximum blood supply. After squaring off the central ends of the flaps, they were turned back and a series of parallel horizontal relaxing incisions was made through the galea aponeurotica, from its deep surface. The flaps were then laid back into place and were easily approximated to each other, obliterating the defect and producing an H-shaped scar (Fig. 1B).

Course. Healing was uneventful except that on the 4th postoperative day the child pulled off her dressing, with slight separation of the wound edges in two places. Neither of these re-

quired secondary suture and she was discharged from the hospital on the 11th postoperative day.

Pathological Report. The diagnosis was "heterotopic brain tissue." Figs. 2 and 3 show the microscopic appearance of the lesion. The paler central area is glial tissue, the folded pattern suggesting malformed cortical gyri and sulci. Under higher magnification, the cells are seen to be astrocytes. No recognizable ganglion cells were observed, although it seems perfectly possible that some may be present in the lesion. The glial tissue is surrounded by well-defined meningeal tissue, with both dural and arachnoidal layers present. The arachnoid layer and the dermis are both abnormally vascular, the appearance being similar to the angiomatous

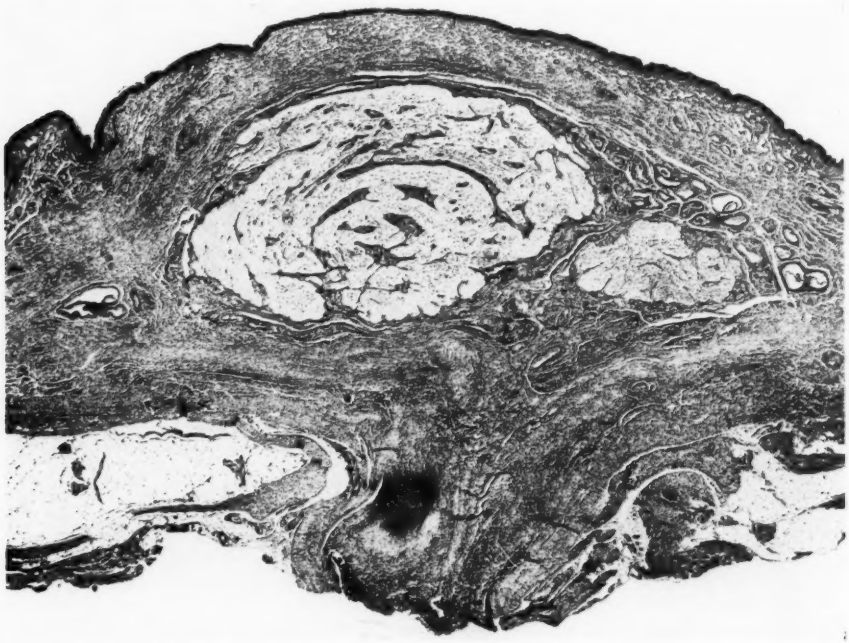

FIG. 2. Section of scalp lesion through meningeal stalk (×9), showing heterotopic glial tissue, with pattern suggesting abortive gyri and sulci. Note that there are no ependymal-lined channels. The laminated vascular arachnoid layer and the outer dural layer of dense collagen can be seen. The overlying scalp contains no skin appendages.

malformation of the same layers seen in many meningoceles and encephaloceles. The meninges converge into a central stalk which was cut across at the level of the pericranium. As previously noted no bony defect was recognized. It seems quite possible, however, that the central meningeal stalk may have penetrated the cranium through a passage so small that it was overlooked.

DISCUSSION

In attempting to explain the embryologic disturbance that produced this congenital defect, a number of questions are raised for which no conclusive answers can be given, but which are of considerable theoretical and practical interest.

The only directly comparable lesion is the apparently misnamed "nasal glioma."

The structure of most nasal gliomas is virtually identical with the anomaly here described and the two lesions differ only in location. Davis[2] critically reviewed the subject and concluded that most nasal gliomas are not neoplastic. He stated that true olfactory bulb tumors do occur but differ in both structure and origin from the more common nasal gliomas which he considered to be "fibroglial heterotopias in the nasal region . . . arising from encephaloceles." The difficulty has been to reconcile the mechanism of their origin with the accepted theories regarding encephaloceles.

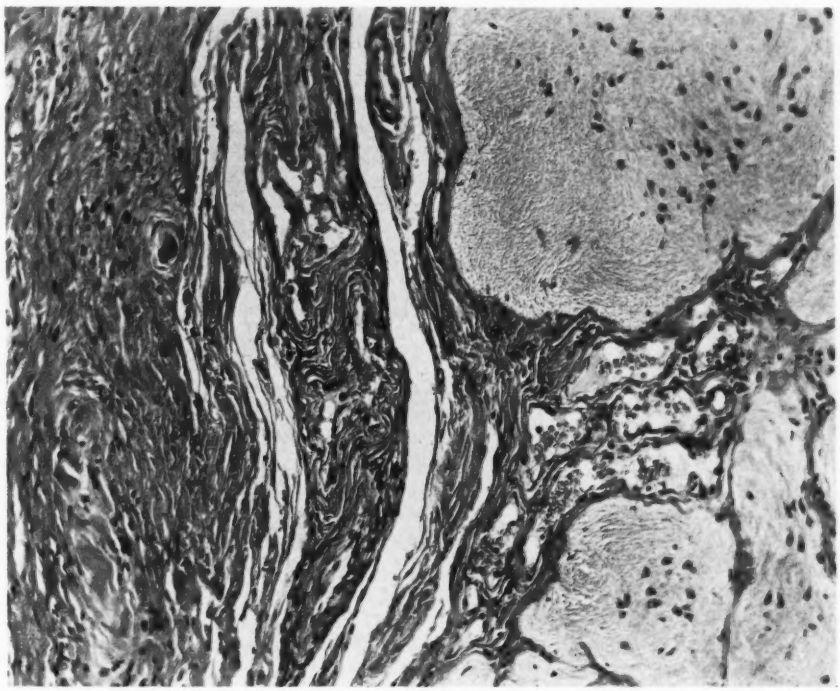

FIG. 3. Section of junction of heterotopic glial tissue and meningeal tissue (×160), showing basic architectural similarity to cerebral cortex, with gyri separated by sulci, and an outer plexiform layer in the nervous tissue. The arachnoid and dural layers of the capsule are easily distinguished.

For many years it was held that encephaloceles resulted from arrested development of the skull which left a defect through which the meninges, or brain and meninges, herniated at a later stage of embryonic development. Recently Patten[5,6,7] presented evidence, based on the study of human embryos, that the herniation theory may not be valid as an explanation for either encephalocele or meningomyelocele. His concept, now gaining wide acceptance among developmental anatomists, indicates that an initial overgrowth of the neural tube prevents the closure of the cranial or spinal covering, the precise reverse of the herniation theory. Such a mechanism would be much more consistent with a rational explanation of both nasal gliomata and of the anomaly described in this report.

As long ago as 1890, Berger[1] pointed out that the structure of the tissue in certain

encephaloceles differed considerably from the structure of the brain to which they were attached. This is in accord with the view that the seemingly extruded brain and covering have actually been "pinched off" at a very early stage of embryonic development by the converging primordial cranial bones and are the cause rather than the result of a cranial defect, when this is present. Stout[8] stated that nasal gliomas occasionally do have a residual connection with the brain, resulting in meningitis when they are removed, constituting additional evidence of their direct relationship to encephaloceles.

If this general hypothesis is accepted, the glial heterotopia over the occiput in this case is the precise counterpart of those occurring over the nose and both lesions represent variants of the familiar encephalocele. These lesions are plainly different from, but apparently embryologically related to, the midline congenital dermal sinuses and the isolated midline cutaneous defects mentioned previously.

In the case of the extracranial glial rests, neuroectoderm seems to have been "left behind," with or without a tract connecting with the central nervous system. Their relatively small size and the absence of a connecting ependymal canal is all that distinguishes them from encephaloceles. Conversely, when a congenital dermal sinus is present, and connects, as it not infrequently does, with an intracortical, cerebellar, or intramedullary dermoid cyst, epithelial ectoderm has been "drawn into" the cranial vault or bony medullary canal. When no connecting tract is present either an isolated congenital glial rest or an isolated dermoid cyst of the brain or spinal cord is the result. The former is, of course, visible at birth while the latter often does not become clinically apparent until advanced adult life.

No clear explanation of the mechanisms involved in the production of these abnormalities is available, but as Matson and Ingraham[4] have stated, " . . . one of the most vulnerable incidents in embryology is the separation of the neuroectoderm from the epithelial ectoderm, which occurs between the third and fifth weeks of intrauterine life. Failure of invagination of the primitive medullary plate to proceed in an orderly and complete fashion, results in a wide variety of defects involving the skin and central neural axis as well as the surrounding structures of mesodermal origin." The remarkable fact is not that these anomalies occur at all but rather that they occur so infrequently.

An equally interesting and unsolved question is why extensive defects such as encephalocele or rachischisis are quite common, while the less severe derangements here considered are extremely unusual. Ingalls,[3] in extensive studies of embryos and term infants, found that gross midline defects, which are relatively frequent at birth, were not commonly seen at the early period of gestation while the small superficial defects were much more frequently apparent at this time.

Another intriguing question is why congenital cutaneous defects of the scalp, though unusual, are relatively common as compared with heterotopic glial rests in the nose or, as in the present case, in the scalp. Although the data on reported cases of isolated midline cutaneous anomalies of the scalp are insufficient to justify a conclusion, it seems possible that the diagnosis in some cases might have been based on sections taken from the periphery. In that event the central glial tissue might have been overlooked. If this is true, cases similar to the present one may be more numerous than the literature indicates. There is nothing in the gross appearance of this lesion that might have distinguished it pre-operatively from a simple cutaneous anomaly.

CONCLUSIONS

The foregoing considerations have considerable practical importance. It seems apparent that congenital cutaneous defects of the scalp, heterotopic glial rests, whether in the nose or the scalp, congenital dermal sinuses, and intracranial or intraspinal dermoids, all belong to the same embryological family as do encephaloceles and myeloceles.

Although such lesions, affecting the scalp, are evidently unusual, they must be viewed with considerable seriousness when they are encountered. Even in the absence of radiological or clinical evidence, the possibility of a direct connection with intracranial structures must be borne in mind. The risk of meningitis after removal of similar lesions in the nose has been recognized. Precautions appropriate to the possibility of intracranial extension must be taken at the time of surgery.

When a congenital defect of the scalp overlies a large blood vessel such as the sagittal sinus, extreme caution is necessary, since the removal of what appears to be a simple encrustation of dried serum may lead to a fatal hemorrhage.

The prognosis following removal of heterotopic glial rests should be excellent. Removal of simple cutaneous defects may carry a somewhat less certain prognosis. If a sinus is visible extending through the cranial bones it should be followed to its termination which is usually in relation to the meninges. It is theoretically possible, however, that an unrecognized extension may be present which could result, in later years, in an intracranial dermoid.

SUMMARY

An unusual, and apparently hitherto undescribed, congenital lesion of the scalp, which proved to be heterotopic brain tissue, has been reported, and its theoretical and clinical implications have been reviewed. It is suggested that similar lesions may actually be somewhat more common than they appear to be, and that all congenital midline lesions of the scalp, however trivial they may seem to be, should be given serious consideration and approached with considerable caution.

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TWO-POINT OR INTERPOLAR COAGULATION

REVIEW AFTER A TWELVE-YEAR PERIOD WITH NOTES ON ADDITION OF A SUCKER TIP

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(Received for publication December 8, 1954)

The essential differences between interpolar and unipolar coagulation are the absence of electrical connection to the patient and concentration of the current, which flows only between the tips of a forceps. Although reported,[1,2] the interpolar method of coagulation has not found universal application. Many neurosurgeons, because of the requirement of greater accuracy in technique, have not taken the time to learn to use it. These are, however, its chief attributes, little damage being done to adjacent tissues and, most important of all, coagulation is not carried along a vessel to deep tissues.

Fourteen years' use has produced convincing evidence that the method is of distinct value, particularly in work around the spinal cord and in the posterior fossa. The addition of a sucker tip (Fig. 1) five years ago has been an aid in keeping the

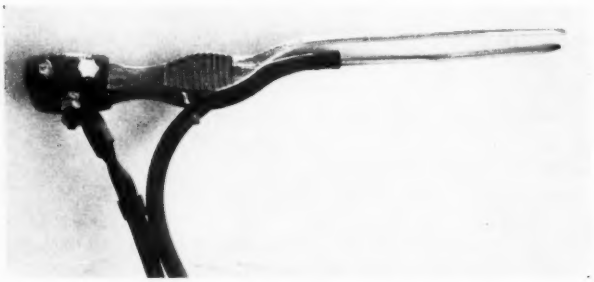

FIG. 1. Two-point coagulation forceps, with sucker tip.

field clear. Bleeding stumps of small vessels find their way easily into the open end in position for coagulation. Advantages of the method include a reduced coagulation current; since the current is concentrated, the total value can be about one-third or one-fourth of that normally used and coagulation may be carried out under a thin layer of cooling saline irrigation. There is little spread of current and a vessel on the surface of the cord or brain stem may be coagulated with relative safety. There need be no fear in coagulating surface vessels of the cord in an area to be incised, as in chordotomy. It is being used consistently for arteries as large as the temporal or occipital, and may be used for veins as large as the external jugular.

The chief disadvantage is the necessity for accuracy in its use, otherwise its limited heat area (between tips only) will not successfully control hemorrhage. In

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addition, the sucker tip, because of its small size, will not take care of a massive hemorrhage and may have to be opened occasionally with an obturator because of plugging or obstruction.

Aside from genera coagulation, the following special applications are felt to be practical:

1. Bloodless cortical incisions can be made with a scalpel after coagulating surface vessels.
2. Coagulation of vessels on the surface of the spinal cord or brain stem (with reduced current).
3. Production of widespread coagulation throughout a vascular tumor by plunging the blades (separated about $\frac{1}{4}$ inch) through the tumor in different directions (current elevated for this maneuver). Removal may then be accomplished similar to that of a less vascular tumor.

Continued use of this method and instrument* has led the writer to call attention again to its advantages. The addition of the sucker tip has added to its usefulness.

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* The two-point coagulation forceps is available through the makers, J. C. Coogler & Son, 7314 Gillen Street, Houston 17, Texas, and also through Codman & Shurtleff, Inc., 104 Brookline Avenue, Boston 15, Massachusetts.

Obituary

OLOF SJÖQVIST

1901–1954

The death of Olof Sjöqvist on December 4, 1954 did not come unexpectedly. Since early June he had been ill with high temperature, severe muscular pain and enlargement of the liver. After biopsy the diagnosis of periarteritis nodosa was established. Under cortisone treatment his condition improved so much that by the end of August he was able to be up and about for short periods, but in October a new attack of the destructive process in his arteries occurred and severe hypertension with eyeground changes and enlargement of the heart developed. His medical advisers regarded his condition as extremely critical, not to say hopeless, but Sjöqvist's indomitable courage and optimism did not desert him. He refused to admit defeat and continued to make plans for future research and for the resumption of his clinical work.

When I visited him for the last time in the end of November I found him in good spirits and in fairly good general condition. We discussed his chapter in the *Handbuch der Neurochirurgie*, which was practically finished when he became ill and I left with his promise to deliver the manuscript ready for print within two weeks. Fate, however, had decided otherwise and on December 4th a sudden attack of auricular fibrillation ended his life within half an hour.

My first acquaintance with Olof Sjöqvist dates back to 1924 when a young and pleasant looking medical student came to me and offered his services as assistant in some experimental work, in which I was at that time engaged. He was accepted and we worked together for several months on problems connected with the sympathetic nervous system. After completion of this work Sjöqvist had to continue his medical studies, and we drifted apart for a while, but he came back in 1932 after some years in general surgery and inquired about the possibilities of a training in neurosurgery and the chances of a neurosurgical career. These the present writer thought to be rather dubious as neurosurgery in Sweden at that time was in its infancy and had only just shed its dependence on general surgery. The creation of a second neurosurgical clinic at that time appeared to be a rather distant proposition and the road to this goal strenuous and long. However, as later events proved, Sjöqvist showed himself to be more clear-sighted than the present writer and upon return from Fulton's laboratory, whence he had gone to round off his clinical training with a year in neurophysiology, he was offered a position as consulting neurosurgeon to St. Erik's Hospital in Stockholm, a position that was soon exchanged for that of surgeon-in-chief to the newly created neurosurgical clinic at the large municipal Söder Hospital in Stockholm.

Olof Sjöqvist was born in Stockholm on December 9, 1901, the son of John Sjöqvist, professor of biochemistry at the Royal Caroline Institute. From his home he brought with him a strong interest in physiology which was further developed during his student years. His physiological training proved to be a valuable asset in his later work as a neurosurgeon and many of his contributions to medical science bear the imprint of his physiological background. Among his published papers, over 80 in number, the most important is his study of pain conduction in the trigeminal root. In this outstanding piece of work he was able to show by fibre analysis that the small-caliber fibres conducting pain and temperature practically all descended into the spinal root of the trigeminus while the coarse fibres conducting sensation of touch turned upwards into the mesencephalic root. Based upon these anatomical facts Sjöqvist devised the operation of medullary tractotomy which bears his name. By this procedure it became possible for the first time to relieve the pain of tic douloureux without the massive anaesthesia of a large portion of the face accom-

panying section of the sensory root. Even though the high frequency of recurrence and a mortality somewhat higher than after root section limited the indications for tractotomy to certain types of facial pain, Sjöqvist's contribution to the surgery of trigeminal pain must be regarded as the most important made after the introduction of root section by Spiller and Frazier in 1901.

Sjöqvist's cheerful personality and ready wit made him a delightful and entertaining companion who easily made friends with both his colleagues and patients.

The Hungarian novelist, Karinthy, in his book *A Journey Round My Skull* describing his experiences as a patient in the neurosurgical clinic in Stockholm, gives a very sympathetic picture of Sjöqvist who was at that time the senior resident surgeon in the clinic.

In Olof Sjöqvist Swedish neurosurgery has lost one of its outstanding figures. He has given much to neurosurgery, and much more could have been expected, had the fruits of his fine mind and wide experience been permitted to ripen. His death leaves us with the poignant feeling of meaningless tragedy, which we all experience, when the life work of a man, still in his prime, is abruptly ended before its completion.

HERBERT OLIVECRONA, M.D.

BOOK REVIEW

PREFRONTAL LEUCOTOMY AND RELATED OPERATIONS: ANATOMICAL ASPECTS OF SUCCESS AND FAILURE. (The Henderson Trust Lectures, Publication No. XVII.) By Alfred Meyer, and Elizabeth Beck. *Edinburgh & London: Oliver & Boyd*, 1954, illus., 60 pp. Price, 10/6.

Dr. Alfred Meyer, Professor of Neuropathology, and his Research Assistant, Miss Elizabeth Beck, both of the Maudsley Hospital of the University of London, here give critical appraisal of their experience with 102 cases of frontal lobotomy in which anatomical data were obtained concerning the extent of the lesion and the nature of the degeneration. It is clear from this series that the radical lobotomy involving lateral structures causes deterioration of personality that may largely offset any beneficial effect that often results from the lobotomy itself. The experience of Meyer and Beck also points to the fact that medial lesions, especially those involving the medial orbital area (that interrupts the thalamo-frontal projection) is often followed by dramatic improvement without intellectual deficit. They find further that cingulectomy is mainly effective in obsessional neuroses but that this limited lesion generally fails to give improvement in the major psychoses.

JOHN F. FULTON, M.D.

NOTICES

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY AWARD

This annual award is to be given to a neurosurgeon either in training or in practice but not having completed his American Board of Neurological Surgery certification, for the best paper on the subject of either clinical or laboratory research completed by the applicant. The submitted papers will be judged by a committee to be named by the Academy.

The award stipulates that the paper shall be presented at the annual meeting of The American Academy of Neurological Surgery by the author, the expenses of the recipient of the award being paid by the Academy. In addition to this there will be an honorarium of $100.

The next meeting of the Academy will be in Hot Springs, Virginia, at the Homestead, October 27 through 29, 1955. Deadline for submission of papers for consideration is August 15, 1955. All papers to be considered should be sent to Dr. Edwin B. Boldrey, University of California Medical Center, San Francisco 22, California, Chairman of the Awards Committee.

AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY

The 9th annual meeting of the American Electroencephalographic Society will take place at the Palmer House, Chicago, Illinois, June 10–12, 1955. Scientific sessions will be held on June 10, 2:00–4:15 P.M. and 8:00–10:15 P.M., and on June 11, 2:00–5:00 P.M.

In addition, there will be two symposia: (i) on Micro-Electrodes, June 11, 9:30 A.M., under the Chairmanship of Dr. Herbert H. Jaspers; (ii) on Clinical EEG Interpretation (Head Injuries), June 12, 9:30 A.M., under chairmanship of Dr. Frederic A. Gibbs.

AMERICAN NEUROLOGICAL ASSOCIATION

The 80th annual meeting of the American Neurological Association will be held at the Palmer House, Chicago, Illinois from June 13–15, 1955.

SYMPOSIUM NEURORADIOLOGICUM

The 4th meeting will be held in London, September 13–17, 1955. Registration will take place on September 12 at the University of London, Malet Street, W.C.1, where the scientific meetings will be held. Radiologists, neurologists and neuro surgeons particularly are welcome.

Subjects for discussion include i) cerebrovascular disease, ii) the localisation of intracranial tumours by means of isotopes, iii) applications of stereotaxometry, iv) new techniques, and v) various papers on neuroradiological subjects. Scientific and technical exhibitions are being arranged.

It is hoped to arrange a joint half-day session with the 2nd International Congress of Neuropathology which is to be held simultaneously in London.

A full social program has been arranged including visits to places of interest for wives and families of members.

Application forms and further information may be obtained from Dr. R. D. Hoare, Secretary, Symposium Neuroradiologicum, The National Hospital for Nervous Diseases, Queen Square, London W.C.1.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

MANUSCRIPTS should be addressed to Dr. Louise Eisenhardt, 333 Cedar Street, New Haven 11, Conn., U.S.A. They should be typewritten in double space, with tabular matter and passages for small print on separate sheets. All materials should be mailed flat.

REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbreviations of names of periodicals should conform to the style used in *A world list of scientific periodicals published in the years 1900–1933* (Oxford University Press, 1934).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

ILLUSTRATIONS. Care should be taken not to use too many illustrations, since the Journal provides no figures free of charge. Color plates also are inserted at the expense of the contributor. If illustrations are larger than 8.5×11 inches, reduced glossy prints should be submitted instead.

SPECIAL TYPOGRAPHY. Tables and also text set up in small print in excess of 10 per cent of composition costs are charged to the author.

PROOF CORRECTIONS will be allowed without charge up to 5 per cent of the original cost of composition. The cost of changes in page proof, if made by the author, will be borne by him. *Page proof* will be submitted to authors only when special questions arise.

REPRINTS. Owing to existing paper shortage, no reprints may be supplied *gratis*. A schedule of charges for reprints is noted on the order forms accompanying the galley proof of each paper.

SUBSCRIPTIONS. The subscription rate for the volume of six bi-monthly issues is $11.00 in the United States and Pan-American Union; $11.50 in Canada; $12.00 in other countries. Single copies are $2.00 postpaid. Single copies of Volume VIII, Number 1, January 1951 issue: A Symposium on Intracranial Vascular Abnormalities: $3.00 postpaid, if available. Volume I, Number 1 is supplied only on orders for the complete volume. Payments may be made in advance to Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.

CONTENTS

1. Cerebral Paragonimiasis. Report of Four Cases. Sun Keun Kim . . . 89

2. The Syndrome of Acute Anterior Spinal Cord Injury. Richard C. Schneider . . . 95

3. Compression Rather than Decompression for Trigeminal Neuralgia. C. Hunter Shelden, Robert H. Pudenz, Donald B. Freshwater, and Benjamin L. Crue . . . 123

4. Aqueduct Stenosis. Clinical Aspects, and Results of Treatment by Ventriculocisternostomy (Torkildsen's Operation). Kenneth W. E. Paine, and Wylie McKissock . . . 127

5. Results of Direct Attack on Nonfistulous Intracranial Aneurysm with Remarks on Statistics. Carl J. Graf . . . 146

6. Cerebral Hemispherectomy for Control of Intractable Convulsive Seizures. L. A. French, D. R. Johnson, I. A. Brown, and F. B. Van Bergen . . . 154

7. Observations on Ventricular and Lumbar Subarachnoid Peritoneal Shunts in Hydrocephalus in Infants. Michael Scott, Henry T. Wycis, Frederick Murtagh, and Victor Reyes . . . 165

Case Reports and Technical Note:

Spinal Extradural Cysts. Report of a Case. Henry M. Cuneo . . . 176

Thrombosis of the Internal Carotid Artery Caused by a Cervical Rib. Ebrahim Samiy . . . 181

Two Cerebellar Tumours with Unusual Features. 1. Cystic Astrocytoma. 2. Papilloma of the Choroid Plexus. R. G. Robinson . . . 183

Posterior Inferior Cerebellar Artery Syndrome Following a Fracture of the Cervical Vertebra. Raplh L. Suechting, and Lyle A. French . . . 187

Heterotopic Brain Tissue as an Isolated Embryonic Rest. C. Marshall Lee, Jr., and Robert L. McLaurin . . . 190

Two-Point or Interpolar Coagulation. Review after a Twelve-Year Period with Notes on Addition of a Sucker Tip. James Greenwood, Jr. . . . 196

Obituary

Olof Sjöqvist . . . 198

Book Review . . . 201

Notices . . . 201

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